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SPINAL INJURIES — A CHALLENGE*

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"After knowledge comes wisdom"—Osler

THE treatment of spinal paralysis has undergone great changes in the past fifteen years. For the past four years in Western Australia all spinal injuries and those patients with spinal paralysis not caused by injury have been treated in a special unit. Such special units, with their teams of workers have resulted in improved management, a reduced death and morbidity rate and an economic saving, for it is no longer "Can a paraplegic ever leave hospital?" but simply, "How long?" Scientific stock-takings are always necessary if advances are to be made and thus this review for the years 1954-58 will set out the results of treatment and some experience gained.

There is now no doubt that all cases of paraplegia are best handled by a specialized unit. During the four-year period under consideration 150 patients with spinal paralysis have been seen—a few for consultation only, but most for management (Table 1). Most cases of injury have been seen within hours of accident and some 65 acute cases have been observed. Of these 34 were quadriplegic, 30 are paraplegic. In the time of review 15 other patients were seen whose traumatic spinal cord injury had occurred a varying number of years before. These presented with complications. Some 16 cases of secondary carcinoma have been treated in the final stage of their illness and 18 cases of poliomyelitis were also encountered (a large number of which occurred during a local epidemic).

The average stay in hospital for acute spinal injuries was one hundred and fifty-eight days, the longest stay being one thousand four hundred and sixty-one days, the shortest forty-two days. The time spent in hospital can now only be reduced when adequate discharge agencies, such as homes and hostels exist. Economically, the provision of a hostel for paraplegics who, though independent, cannot go to their own homes would be sound. During each month that such patients remain in hospital awaiting final discharge this State pays one hundred and fifty-four pounds ten shillings (£154/10/-).

Most of this review is concerned with the 65 cases seen within a few hours of injury and considers the early surgical management of these cases. Figs. I and II show incidence of traumatic cases, and their levels.

EARLY ADMISSION

It has been repeatedly observed that those patients admitted within a few hours of injury are always more easy to manage than those whose admission, for one reason or another, has been delayed for days or even weeks. This can be attributed to:

(a) Early accurate diagnosis and thus attempt at prognosis.

(b) Efficient ancillary methods such as nursing and physiotherapy.

* From Spinal Unit, Royal Perth Hospital. Based on a lecture delivered to Annual Scientific Meeting, Royal Australasian College of Surgeons, Perth. August, 1958.

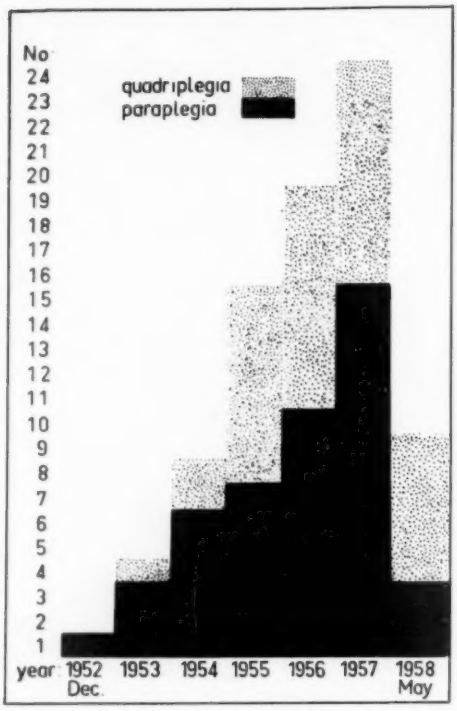

FIG. I. Traumatic cord injuries.

Accurate diagnosis as soon as possible into incomplete or complete lesions is of fundamental importance. This in the quadriplegic where spinal shock frequently takes some time to disappear may be difficult. Meticulous clinical examination can never be replaced by special examinations. At the Royal Perth Hospital Spinal Unit there is a double check by both the author and his neurological colleague, both of whom see all cases as they are admitted.

In the time under review 28 incomplete and 6 complete cases of traumatic quadriplegia were seen; this estimation being made after spinal shock has disappeared. For traumatic paraplegia the figures were 16 incomplete and 30 complete lesions (Table 2). Diagnosis may be difficult until spinal shock has passed off. Spinal concussion disappears quickly, usually under twelve hours, whilst spinal shock particularly in quadriplegia is slow to abate and thus the accurate neurological position can be masked for varying periods of time. A variable factor, this has been observed to last five weeks, the patient (a quadriplegic) remaining atonic with absent reflexes, although finally making a very good, though incomplete, recovery. Repeated clinical examinations may be necessary, even two in one day, to follow the neurological changes.

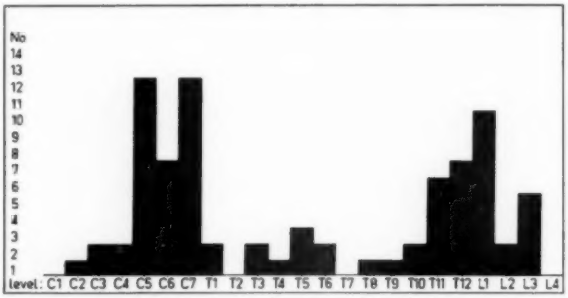

FIG. II. Levels of traumatic lesions.

Patients admitted forty-eight hours after accident all had an obstructive bronchitis and pneumonitis, their bladders were already infected and a few had early pressure sores. On the other hand all cases admitted within twelve hours, despite long distances travelled (up to 500 miles) had no pneumonitic problems and some had sterile urines for the first three to four weeks.

This attempt at accurate diagnosis is the most important early method of management. Emphasis on this must continue for too often an incomplete lesion is missed. Having decided on the completeness or not at first or second examination then a prognosis may be formulated for anxious relatives.

Paraplegics whose lesions are complete for six to eight hours after accident will remain

so. Accurate neurological and bony assessment is of vital importance. Holdsworth (1954, 1956) points out that frequently in lumbo-dorsal injuries without this proper and correct correlation cord lesions can be assumed that are in reality root lesions. A glance at the anatomical chart (Fig. III) will remind one of this fact. Any neurological sign, motor or sensory, despite being very perverted, is a sign of an incomplete lesion and must be searched for. Slow recovery of root lesions can be anticipated, in some cases taking up to years to be effective. For quadriplegia accurate early diagnosis is not so easy and the prognosis is not so grave. Of the 34 quadriplegics seen 24 were flexion injuries, 7 were extension injuries, one was a gunshot injury and two were uncertain of etiology. Bors (1956) in a discussion of quadriplegia noted that 66 per cent. of their 233 cases were incomplete, so that two of every three cases admitted to any hospital may prove to be so with improved prognosis.

Immediately after admission, it is important that patients come under the management of a proper team, for such teamwork has been proven to be essential in the management of spinal paralysis. Our "team" consists of orthopaedic, urological and neurological colleagues, of nurses, of physiotherapists, occupational therapists and dietitians with, finally, a social worker and secretary. Each member has their important part to play and each must co-operate and assist in one another's activities. There must be no hard and fast line across which one member must not trespass. The Medical Director whether orthopaedist, neurologist, urologist or physician, must act as the co-ordinator of all treatment and activities. I am certain that the patient's

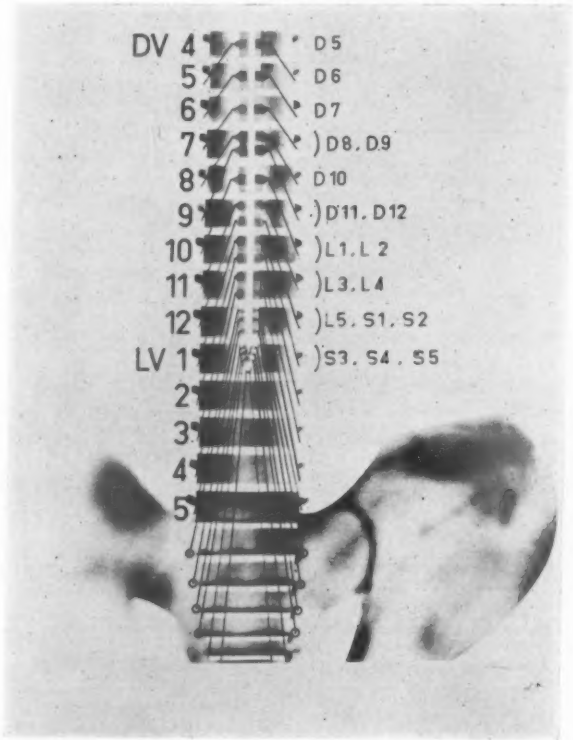

FIG. III. Diagrammatic relations of spinal cord and cauda equina to the spinal column.

TABLE 1

CAUSES OF SPINAL CORD LESIONS

Cause	1952	1953	1954	1955	1956	1957	1958	Total
Traumatic—acute	1	3	6	13	18	17	7	65
Traumatic—late admission	–	1	2	2	1	7	2	15
Poliomyelitis	–	–	3	3	9	3	—	18
Secondary carcinoma	–	–	2	6	1	3	4	16
Spinal cord tumours	–	–	1	1	3	1	1	7
Miscellaneous	–	2	—	5	12	9	1	29
Haemorrhage								
Myelitis								
Spina bifida								
Spastic								
Spondylitis								
TOTAL No.	1	6	14	30	44	40	15	150

life depends on such co-ordination. I have frequently seen patients admitted with acute paraplegia, whose life has been saved by the devoted attention in the first forty-eight hours of a physiotherapist or members of the nursing staff. In the early stages these two members play an intensively important part in the proper management of spinal paralysis. Spinal injuries issue a challenge to medical ancillaries and a field where physiotherapy, for example, has made much advance.

EXPERIENCES IN SURGICAL MANAGEMENT

The relation between bony injury and neural damage is an irregular one so that neural sparing can be found with severe bony displacement particularly in the cervical region. Conversely minimal bone damage can be associated with severe irrecoverable neural damage.

During the review 39 patients were studied who had been subjected to open surgery on the spinal column. Of these 32 were operated upon the first two weeks after admission and seven at varying periods after this time; Fig. IV illustrates these cases. In the early stages of our organized care the author is certain that operations were carried out on some who could not be helped by surgery, and on whom now surgery would not be advised. Laminectomy and open operation have uncommonly proved of benefit. Fourteen of these cases were so explored to help correct the position of the fracture and were judged as needing internal fixation. Of these 10 had fixation at the lumbo-dorsal junction, three in the cervical area and one in the mid-thoracic region. Three other lumbo-dorsal lesions were explored and found impossible to fix because of fractures of spinous processes. Of those fixed in the lumbar area only two were satisfactory. Another two whose

TABLE 2

SHOWS INCIDENCE OF COMPLETE AND INCOMPLETE LESIONS IN TRAUMATIC SPINAL PARALYSIS

Type	1952	1953	1954	1955	1956	1957	1958	Total
Paraplegic								
Complete	1	3	4	5	5	10	2	30
Incomplete or below level L1 with root escape			2	2	5	5	2	16
	1	3	6	7	10	15	4	46
Quadriplegia								
Complete				2	2	2		6
Incomplete		1	2	6	7	7	5	28
		1	2	8	9	9	5	34
Total No. of cases	1	4	8	15	19	24	9	80

Number of quadriplegics complete on admission who showed useful recovery ... ... 12 — 35 per cent.

Number of paraplegics (above L1 segment) complete on admission who showed useful recovery ... ... 2 — 4 per cent.

ixation proved satisfactory died ten and two lays post-operatively from a cerebral embolus nd the severity of other injuries. The remaining six had their plates removed for pain, or cutting through skin and for obvious oosening. The others who were explored in arly stages were so for a variety of reasons see Fig. IV).

L2 and 3 (lesion at L1, 2) following an extensive removal of the facet articulations from T12-L4 and a wide laminectomy.

During the review a further 41 cases were studied where early operation was not advised except for skull traction in flexion injuries of the cervical spine. In 9 out of these

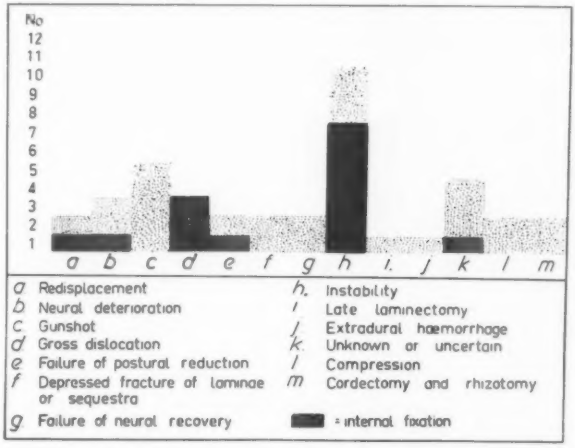

FIG. IV. Reasons for open operation on the spinal column.

Four patients (10 per cent.), three cervical lesions and one lumbo-dorsal injury, were encountered in whom thorough clinical review has made me decide that operation had indeed caused some deterioration in the spinal injury. In three of these cases recovery occurred, and the patients eventually returned to their pre-operative condition or later improved, an improvement that would probably have occurred without operation, since it did so after an interval of two months. In another the patient did not improve and he has been left with incomplete paralysis.

Whenever surgery is considered the surgeon must consider whether such may indeed make the patient worse. Here are four examples in which surgery was harmful to the patients and where a more conservative approach would have left the patient immeasurably better off. I am sure some of the deterioration observed was due to spinal cord handling. Skeletally dysfunction can be severe after operation. I have reviewed one case operated on eight years ago for a cauda equina lesion where a neurotrophic joint now exists between

cases operative interference would in the early years of our work have been advised but for various reasons such as severe head injury, long delay before admission, severe hypertension with urinary insufficiency, operation was not carried out.

Some of these cases were equally if not more severely injured than those operated upon in the earlier period of our observations.

In consideration of the surgical methods all are agreed that cervical dislocations with or without fracture are best treated by skull traction (Crutchfields forceps) and reduction by weight extension. This may in itself be sufficient to reduce the manometric block and allow cord recovery. In fact, in our opinion, other procedures are very rarely indicated in the cervical column. Cervical redislocation with instability is an indication for internal fixation and bone grafting.

Elsewhere the common method used will be postural reduction of the fracture or fracture dislocation unless special circumstances occur. The use of pillows or packs will allow

such to be carried out without great difficulty. No extra harm will result provided all the staff realise the great gentleness necessary in turning and avoidance at all times of rotation at the fracture site. In the thoracic region pain and gross dislocation can be the only important reasons for early or late operation. Laminectomy must be reserved for those rare cases where such will relieve symptoms and signs of a structural compression. Thrombosis and oedema are always difficult to be certain of and do not necessitate open operation.

FIG. V. Severe fraction dislocation L2 on L3 necessitating open reduction.

What then are the indications to operate on patients with acute traumatic spinal paralysis?

(1) Neurological disturbances

(a) Incomplete cases which become complete with spinal block. A minimum procedure will be all that is permissible, certainly no cord handling.

(b) Cases with complete spinal paralysis with no bony injury and a complete spinal block.

(c) Cases with severe root pain and increasing paresis.

(2) Skeletal

(a) Severe irreducible fractures and dislocations (Fig. V).

(b) Where quite irrefutable evidence exists of disc or bony sequestra causing compression.

(c) In cases of "unstable" fractures not maintainable by conservative means, internal fixation is useful.

(3) Psychological reasons

In this series one patient requested open operation for psychological reasons, stating that he wished to satisfy himself that exploration would not assist.

Trying to reduce these variables to a common denominator has proved difficult, for each case must be carefully assessed. One must make final judgment after considering all factors. If in doubt no operation is advised. Operative trauma, no matter how delicate, may be too much for an almost totally damaged cord, already damaged by direct trauma or by interference with its blood supply at the time of accident.

RECOVERY AND ASSESSMENT OF OPERATIVE TREATMENT

(See Table 3)

The author has not seen a paraplegic complete (I repeat complete) on admission six to eight hours after accident recover. One paraplegic has been encountered who recovered after exploration for a suspected extradural haemorrhage and another recovered following operative reduction of a fracture dislocation having deteriorated prior to admission. Even without this operative interference recovery may have occurred in the latter.

Rarely in quadriplegia does open interference help a patient to recover. In one case in this series a severe laminal fracture thought to be causing compression clinically was encountered. At operation the lamina was found to be quite loose but not causing severe compression. Two quadriplegics were operated upon early after admission because of neural deterioration; neither recovered because of the surgical procedure. This deterioration is not uncommon in quadriplegics and is more commonly due to oedema or thrombosis. We have observed that of 34 quadriplegics 35 per

cent. (12 cases) made useful recoveries. Of these 12, three were originally diagnosed as complete and nine incomplete although severe even on admission. This figure has excluded that group of three who were quadriplegic for very short periods up to six or eight hours and thus were usually only suffering from spinal concussion. The use of the term "complete" should rarely be used until spinal shock has disappeared.

not transferred to the Spinal Unit. Thus the need for observation at the initial place of admission.

I am now certain that all incomplete lesions should be treated without open operation despite severe displacement which is rarely an indication for such. The risk of making these complete even with extraordinarily careful surgical technique is considerable

TABLE 3

COMPARISON BETWEEN OPERATIVE AND CONSERVATIVE EARLY MANAGEMENT

	Operation		*No Operation*	
Quadriplegia	Total	10 (5 cases due to bony problems)	Total	24
	Recovery	2	Recovery	13
Paraplegia	Lumbo Dorsal	18	Lumbo Dorsal	11
	Others	6	Others	7
	Total	24	Total	18
	Recovery	3 (Two may not have been due to operation; of these one was incomplete before operation.)	Recovery	0
	Root escape	9 (L.D. only)	Root escape	7 (L.D. only)

Spinal concussion as distinct from spinal shock is transitory and usually has disappeared before patients are admitted. It is significant, however, that in all cases even the most transitory show some neurological aberration months after injury, usually only minor signs such as incomplete recovery of temperature sense or ataxia of spinal origin. Of the 34 cases of quadriplegia six proved to be quite complete, thus 33 per cent. of cases were incomplete. Of the 24 flexion injuries, only 10 showed useful recovery, of the extension two (Table 8). I do not believe these figures to be sufficient to prove that any difference in prognosis exists in the two groups. These results are not dissimilar to the figures of Bors (1956) who noted 66 per cent. to be so, for his figures exclude all those who showed useful recovery at the initial place of hospitalisation and thus were

(four cases out of 39 — 10 per cent.). In complete lesions similar conservatism must be used with surgical judgment. In the cervical region this has been proven correct, not one of our six cervical explorations resulted in recovery, three showed deterioration. In the lumbo-dorsal region I have not convinced myself, except in one case where a large bony sequestrum was present in the spinal canal, that any operation I have performed further protected the cauda equina from damage. Careful nursing was of much greater importance. I still have some doubt as to the use of operative reduction in cases of dislocation with locked facets particularly in the lumbar region where the canal capacity is greatest. The maximum damage occurs at the time of accident, X-rays seen at the time of admission will, because of muscle spasm and muscle splinting (a very efficient mechanism)

show the fracture in a partially reduced position. Thus in the lumbar spine at least the cauda equina will not be on stretch unless further severe damage occurs in hospital.

TABLE 4

RECOVERY IN TRAUMATIC CASES

Type	No.	*Cause*	*Useful Recovery*
Quadriplegia	24	Flexion injuries	10
	7	Extension injuries	2
	2	Type of injury uncertain	Nil
	1	Gunshot wound	Nil
	34		12
Paraplegia	46	Flexion injuries	3 16 (with significant root escape)

Considering internal fixation at the lumbo-dorsal junction in only two of ten cases was this satisfactory and helped in nursing. Three cases were found impossible to fix and nine others were treated where for other reasons then instability internal fixation was not advised. Not one of these 12 cases proved difficult to manage by usual methods and only one had severe pain which was causalgic in type. This is not uncommon in incomplete lesions. One of the internally fixed cases had similar pain and all cases with and without internal fixation showed degrees of vertebral collapse three months after accident. No comments on the use of laminectomy from this series can be made. Except in the cervical column where bone grafting is of use, it is my opinion that lumbar bone grafting is rarely indicated for stability will be obtained without. I have repeatedly observed that these spines stabilize within the first few weeks after injury.

In a comparison of recovery with and without laminectomy by Bors (1956) the overall improvement was greatest in both quadriplegia and paraplegia when no operative interference was deemed necessary. It is possible that those cases managed with operation were, however, more severe. Commarr (1956) in an unpublished report states that very few cases where laminectomy has been performed have recovered because of their operative treatment. Certainly many patients with spinal block are needlessly subjected to laminectomy. Spinal block has not proved to be an absolute indication for operation. It was significant, however, that in both reviews by Commarr (1958) and Commarr and Kaufman (1956) that no patient with complete block and without laminectomy recovered. A further significant observation showed that only quadriplegics who had been treated conservatively showed full recovery. Manometry is only one factor to be considered. Guttman (1953) states that manometry is quite unable to differentiate between oedemal obstruction and that caused by skeletal obstruction. Commarr and Kaufman (1956) state that laminectomy after two to three weeks and during the first month may even improve the prognosis as cases due only to oedema will thus be eliminated. Despite the work of Schneider (1951, 1955), who maintains that cases of acute anterior spinal cord syndrome following injury should always be operatively relieved, I do not believe that early surgery is indicated for such. In this series, four such cases were encountered, all of whom showed useful recovery without operation.

Most writers agree that in spinal injuries:

(1) Deterioration of neural function in the early stages is an indication for a decompressive laminectomy, when the patient is admitted as an incomplete lesion and on becoming complete.

(2) That spinal block without bony damage is also an indication for operative decompression.

And there the agreement ends.

Others have noticed the above peculiarity of cervical cord injuries (Guttman, Commarr, Dewar, Jouse, Holdsworth and Sutcliffe-Kerr (personal communications)). It may be concluded that:—

(a) Dorsal lesions are more commonly complete and remain so.

(b) That cervical cord lesions show very great discrepancy between cord lesions and bony damage. Such lesions show a phenomenon of cord sparing and root recovery.

(c) That lumbo-dorsal lesions commonly show root recovery long periods after injury where even two segments recovery is of great significance.

All agree that the neural canal anatomical relations both as to capacity of spinal canal, size of cord, configuration of facets and alterations in relation to type of anatomical build are factors, which must be taken into consideration as well as severity of injury. The latter is a difficult factor to evaluate; it is possible that most of the cases who survive are in a less severe group.

The author feels that only as experience mounts with properly observed and recorded cases can these questions be answered.

Unfortunately, as is so frequently found, in such studies hasty decisions have resulted in unnecessary operative interference. Many cases, recorded and unrecorded, have been made worse by surgery.

Finally it can be concluded that in traumatic cord injuries that there are very few indications for operative interference. The answer to this perplexing question of indications for operation is not yet solved. From the recorded observations a further conclusion can be arrived at; that no hasty decisions need be made, for time is an important method of clinical evaluation. Only as recorded series of cases mount which have been evaluated properly from the date of early admission can a clearer picture emerge. The individual observation must be merged with large series all investigated and watched in the same way. Perhaps in Australia a combined approach by Spinal Units might be made to this problem.

UROLOGICAL MANAGEMENT

The author freely admits, after a study of 150 cases in many phases of their management, that the paralyzed bladder offers to all of us a challenge that needs the attention of urologist, orthopaedist and all those interested in spinal paralysis.

Working from a knowledge of simple physiology the human bladder has but two functions—that of storage, and that of emptying—the latter being, as is expressed politically at present, "a balance of power" between the detrusor and the sphincter mechanisms. All patients are first treated with an indwelling Foley's number sixteen catheter, which is clipped for a regular period of two hours daily to ensure a good bladder capacity and drained into a sterile bedside bottle. Eighteen months ago we gave intermittent catheterization a trial on four patients, but found it in our hands to be unsatisfactory for:

(a) Bladder infection occurred within forty-eight hours.

(b) Severe urethritis soon supervened.

(c) Debris soon mounted up and made washouts necessary.

Our two aims in treatment are:—

(1) To prevent infection and other bladder complications.

(2) To get rid of the catheter as soon as possible.

From our experience we can say:—

(i) That mobilization, with standing, walking and chair activities is the most important single factor in overcoming infection, reducing residual urine, preventing ureteral reflux and stone formation and aiding bladder emptying.

(ii) That until spinal units are able to operate in separate wards infection will remain at 100 per cent.

(iii) That transurethral bladder neck resections are most useful in the lower motor neurone or cauda equina cases.

(iv) That spinal alcohol block may not only help spasm, but also increase the bladder capacity in a tight spastic organ.

(v) That, unless absolutely indicated, as in one case for a traumatized urethra, suprapubic cystostomy must be avoided. In our experience suprapubic cystostomy was performed only three times in three cases. We are now agreed that in two of those cases transurethral drainage should have been used.

Of our 150 cases, 91 are available for study, 44 now being "normal" and 15 deceased. Bladder infection is universal. In three cases death occurred from renal failure. We have seen no cases of perineal fistulae.

In complete cases the catheter was dispensed with as soon as possible, the shortest period being three weeks, the longest being many months.

We are certain that proper catheter drainage is the best method of treating recurrent infections, and hydronephrosis with or without reflex. Pyrexia is frequently caused by urinary tract obstruction and retention, not by infection. We are equally certain that allowing an overflow retention to develop or managing the acute neurogenic bladder by expression is incorrect, for continued distension will cause permanent damage and prevent intrinsic mechanisms from being efficient. Such a method also allows residual urine to bank up. Proper follow up regularly and consistently is teaching us much of importance in maintaining these patients well urologically. Of 91 cases 30 have reflex bladders, 12 have automatic bladders, 5 are incontinent, 39 are unclassifiable, having good bladder awareness and precipitate micturition, 2 are unknown and 3 have permanent catheters—these are females.

EXPERIENCE WITH COMPLICATIONS

Bedsores

Ancillary treatments reach their zenith with proper nursing care associated with physiotherapy and occupational therapy. The incidence of such will rapidly disappear with specialized care in spinal units. In this unit with 65 acute admissions, one serious and four minor pressure sores have occurred in the acute states of the illness. All patients and staff are taught to have an acute awareness of this problem. In the same time 18 other patients, traumatic and non-traumatic, were admitted with sores, either from other wards or from outside the hospital. One is continually surprised at difficulties expressed by staff at the impossibility of preventing such sores. The above figures speak for themselves. Pressure sores need not occur. The patients must also be taught their responsibilities in their prevention. Of all the sores seen (26), four were very large, sacral or trochanteric, taking up to one year to heal, 14 were small and healed in a few weeks, and 3 were heel sores, which usually heal quickly, but always give some disability through loss of subcutaneous tissue. Finally, 3 were of the difficult sinus type associated with bursae over sacral or great femoral tuberosities, 1 was self inflicted and 1 was over the anterior tibial surface. Many factors are involved in the proper management of these conditions —anaemia, renal infection, proper mobilization of scars, dietary deficiencies, psychological problems, local methods of eradication by suture, flaps, split-skin grafts, and excision may be needed. Of all the factors the removal of pressure must still be emphasised as the most fundamental and important, aided by proper nursing techniques of posture. All our patients admitted with sores were anaemic with haemoglobin values ranging as low as 10 gms. per cent. In paraplegia a haemoglobin under 13.6 gms. per cent. must be regarded as necessitating treatment. All traumatic cases and most others have received blood transfusions, even in the absence of common contributory factors such as renal infection, protein loss and loss of sympathetic tone. Factors such as red cell volume, plasma volume and red cell survival may be important in such anaemias. These are at present under investigation.

Contractures

Joint contractures occur in untreated spinal cord injuries but should rarely be seen in those who have adequate physiotherapy from the commencement of their hospitalization. Two severe cases of contracture occurred in the 65 acute cases, the first in the elbows of a quadriplegic and the second in a paraplegic who also had an old lower motor neurone disease. Minor contractures tend to occur very easily in the knee with the shortening of the quadriceps. Proper posture in the extended position of all joints and the careful observance of a full range of movement of all joints will prevent most contractures. Attention to detail is the only way in which ankle contracture, e.g., equinous deformity, can be overcome. All paralysed joints must be put through a full range of movement at least once a day and the patient instructed by individual or by class activity to maintain such ranges of movements. Three other patients were admitted to this unit with severe contracture of some years duration. In two, hip contractures were relieved by bilateral Soutar's operation and in two the equinous deformities were relieved by triple arthrodesis.

Spasm

The development of spasm must be anticipated in all quadriplegics and in those paraplegics who may have some isolated cord function. Most spasms are flexor in type; we have only observed three cases where the extensors remain in permanent spasm. The proper posture in the extended position of joints and the maintenance of a proper range

of passive movements will stop any shortening of the flexor muscles and thus prevent or minimize, by means of postural reflexes, the onset of spasm. Treatment will depend on the severity of the spasm, but no operative procedures must be considered until a trial of the physiotherapy methods of extension, mobility, passive manipulation and stretching have been tried. Severe irreducible spasms which are interfering with the mobility and indeed with life itself, may be treated by:

(1) Neurectomies.

(2) Tendon lengthenings (a lengthening of the tendo achilles will often reduce general spasm).

(3) Tentomies—usually of transitory use only.

(4) Alcohol block—for severe and otherwise complete uncontrollable spasm.

TABLE 5

SPINAL PARALYSIS — TRAUMATIC AND NON-TRAUMATIC RESULTS OBTAINED

Result	*Traumatic*	*Non Traumatic*
Deaths—acute	9	
One death excluded (died within minutes of admission)		
Deaths—chronic	3	16
not caused by spinal lesion	1	1
Hospital inpatients	20	13
Aged and severely disabled	2	3
Not working	10	8
Total recovery and working	6	1
Complete lesion—working or in training	15	20
At school	1	8
Incomplete lesion—working or in training	13	—
	80	70

Percentage of traumatic spinal cases working (excluding deaths, hospital cases and complete recoveries):

Working — 60.5 per cent.

Not working — 39.5 per cent.

Percentage of deaths in acute state — 11.25 per cent.

Percentage of non-traumatic spinal cases working (excluding deaths, hospital cases and complete recoveries, aged and children at school):

Working — 87.7 per cent.

Not working — 22.2 per cent.

Alcohol block has been used five times in the group of patients under review. We have no extensive experience with rhizotomies, having had this performed in one case only. This case later had an alcohol block for recurrence of spasm. Some spasm recurs after alcohol block over a long period of time, but is never severe and rarely sufficient to warrant a second alcohol block.

Spasm may be responsible for unusual complications such as stress fractures and two cases of stress fracture of the femur have been observed due to severe and uncon trollable spasm.

SURVIVAL

The author was fortunate in being able to study the records of some paraplegics who had been treated in a Spinal Unit set up at Hollywood General Hospital during the 1939-45 war.

Eight patient histories were examined; two of the patients the author has seen regularly for follow-up and are traumatic lesions included in the above review. All of these patients have now survived varying periods from forty years to twelve years after their accident. There were two incomplete lesions and these patients remain well, despite having had a suprapubic cystostomy and multiple complications such as spasticity, contracture and bed sores. The other four included one patient with tuberculosis, one patient with spinal cord tumour, one case due to trauma and one due to granuloma. All these patients showed renal infection and had contractures and bed sores prior to treatment at the Repatriation General Hospital. These paraplegics required up to two years to be adequately rehabilitated. In one patient not examined for three years the author was surprised to find that neurological sensory recovery had occurred two and a half years after the accident, a manifestation of a lower motor neurone lesion, the patient having been discharged from hospital with no sensory recovery below L1. On being seen three years later he had adequate sensory recovery including S1 and 2. In the follow up of our own patients I have noticed on two occasions the development of spasm in an otherwise flaccid lesion, two years after injury; this is felt to be a manifestation of peripheral nerve regeneration.

As to survival we have not had a sufficient follow up time but our experience has led us to make a few important observations:

(a) Because of the universal renal infection and morbidity we consider that ordinary longevity is not completely certain.

(b) Those who maintain an active home treatment programme of standing, regular micturition and bowel habit, regular drinking, will usually remain well.

(c) Relaxation of such a programme will give rise to all manner of complications.

(d) Those who have been rehabilitated by the definite programme now available, have maintained good general health and have been able to remain at work consistently.

In consideration of work activity in acute traumatic spinal injuries, the eight cases seen can be resolved as in Table 5. These figures show that 28 of 39 disabled patients are working. Guttmann (1954, 1957) in his review of 1,000 paraplegics, notes that 69 per cent. are working full time in those cases discharged. An improvement in the above figures as stated can be expected as our methods of rehabilitation improve.

CONCLUSIONS

(1) A review of 65 acute spinal injuries is presented.

(2) The importance of accurate clinical examination and assessment is stressed.

(3) Complete paraplegics will rarely recover. Thirty per cent. of severe quadriplegics may recover.

(4) Operation is rarely indicated.

(5) Bladder management needs careful assessment to prevent infection and improve bladder emptying. Satisfactory bladder function can always be expected.

(6) Normal survival is not yet proven. Some 60 per cent. in this series are working.

ACKNOWLEDGEMENTS

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THE RELATIONSHIP OF SOLAR RADIATION TO MELANOBLASTOMA

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IN Australia there is an increasing incidence of melanoblastoma of the skin as one travels north towards the equator. Taking the latitude of the capital as representing each state, Tasmania, at 43° south, has the death rate of 8 per million for this type of cancer, while in Queensland, at 27° south, the rate is 23 per million. Sydney, at 34° south, occupies an intermediate position nwith 15 deaths per million (Lancaster, 1956). In addition to this geographical variation, Lancaster and Nelson (1957) were able to correlate lack of skin pigmentation and exposure to sunlight with the incidence of melanoblastoma.

Effect of Solar Radiation upon the Skin

Exposure to sunlight causes a characteristic change which is seen most frequently in fair-skinned persons. In the early stages there is a simple mottling of pinhead sized yellowish areas in an otherwise normal skin, best seen on the forehead or side of the neck. Later the skin loses its elasticity, becomes thicker with deeper furrows, shows irregular pigment spots and often becomes erythematous (Fig. I).

Histologically there is a progressive degradation of the structures of the subpapillary and papillary zones of the dermis. At first collagen in the region of the subpapillary plexus of vessels is replaced by a mat of elastic fibrils. The vessels in the affected areas are dilated and prominent. Gradually the condition encroaches on the papillary layer of the dermis, and at the same time or preceding it, the vertical capillary loops are found to be dilated. As the process advances towards the epidermis, the zone of elastosis gradually becomes homogenized and the overlying epidermis may undergo atrophy. In an advanced lesion there is atrophy of the epidermis with increased pigmentation and in the papillary and subpapillary zones of the dermis, collagen is replaced by a homogenous material which is not readily stained by ordinary histological methods; vessels in the area are ectatic and fibroblasts are prominent and atypical. In our opinion, these dermal changes are due to altered tissue metabolism originating from injury to the dermal vessels by ultra-violet light (Figs. II-IV).

FIG. I. Woman aged 74 years. There is severe collagen degeneration with epidermal atrophy. The pigmented lesion had been present for nine years and the malignant nodule for one year.

Because the vessels of the subpapillary zone lie parallel to the skin surface they are vulnerable to any ultra-violet light penetrating to that depth. The vertical loops, though approaching closer to the skin surface present a smaller target. We believe that the ectasia which is seen first in the subpapillary plexus of vessels and only at a later stage in the vertical capillary loops, is a consequence of this anatomical arrangement. Collagen degeneration starts around the vessels of the subpapillary plexus and later spreads up into

the papillary zone, eventually replacing all normal collagen. These facts suggest that the effect of ultra-violet light on the blood vessels is to impair the metabolism of the surrounding connective tissue and this is probably accomplished by a change in the ground substance. Whether elastin fibrils are manufactured from abnormal ground substance *de novo*, or whether in its presence collagen becomes transformed into elastin, has not been determined. Whatever the mechanism, elastosis is only a phase in the evolution of the disorder. In the final stage fibroblasts are manifestly atypical and the production of fibrils has ceased.

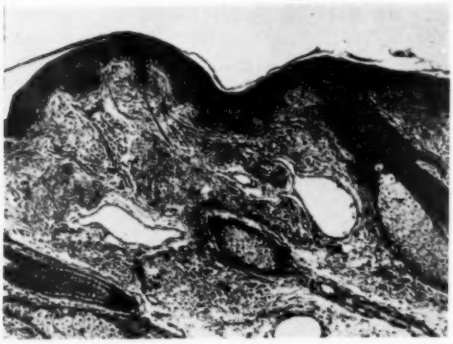

FIG. II. Male aged 68 years; skin of ear. Ectatic dermal vessels surrounded by elastosis adjacent to an acquired melanoblastoma. Haematoxylin and eosin (x 150).

In Great Britain and in the United States this condition is known as "senile elastosis." We prefer the name "collagen degeneration" because elastosis is merely a stage in the evolution of the condition, and because the condition is not confined to the elderly. We often see collagen degeneration in patients in their twenties and sometimes in very fair persons in their 'teens. The age of onset seems to be determined simply by the cumulative effect of solar exposure and the lack of epidermal pigment. It is therefore more marked in fair than olive skinned persons and occurs mainly in those who work or play in the sun.

Collagen degeneration must be differentiated from colloid degeneration of the skin as has been done so clearly by Percival and Duthie (1948). This condition is partly caused by exposure to sunlight and its presence therefore reflects a biological reaction to ultra-violet light.

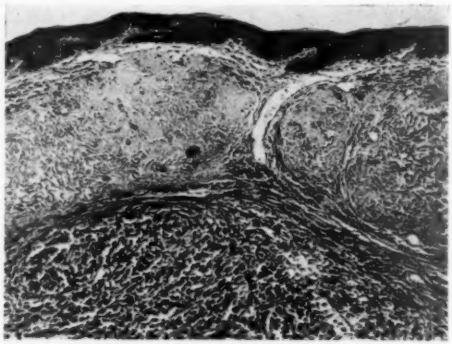

FIG. III. Female aged 66 years; skin of face. The elastotic dermis is becoming homogenized. Melanoblastoma from an acquired lesion nearby is infiltrating the deeper portions of the dermis. Haematoxylin and eosin (x 75).

In our previous paper (Mackie and McGovern, 1958) we correlated the presence of collagen degeneration of the dermis with the development of solar keratoses, keratoacanthoma, squamous cell carcinoma and basal cell carcinoma. We concluded that collagen degeneration of the dermis profoundly affects the nutrition of the epidermis thereby causing dyskeratoses and promoting the development of carcinoma.

TABLE 1

INCIDENCE OF MELANOBLASTOMA ACCORDING TO SITE

	Male	*Female*
Head and neck	26 (33%)	44 (34%)
Upper limb	12 (15%)	22 (17%)
Trunk	24 (30%)	16 (12%)
Lower limb	16 (20%)	46 (35%)
Mucosae	2 (2%)	2 (2%)
	80	130

ANATOMICAL DISTRIBUTION OF LESIONS

Between July, 1950, and December, 1957, biopsy specimens were received from 229 patients with primary melanoblastoma of the skin and mucosae. The sites of these tumours

re known in 210 cases and they are set out n Table. 1. In the case of patients with more han one primary growth, only the initial esion has been classified.

The outstanding feature of Table 1 is the igh incidence of melanoblastoma in the head nd neck area in each sex, the trunk in the ıale and the lower limb in the female.

Again, where there has been more than one primary growth, only the first lesion has been classified.

The main fact illustrated in Table 2 is that on the average melanoblastoma occurred earlier in moles of the congenital variety than in those originating in adult life. Except for acquired lesions of the head and neck, melano-

TABLE 2

)IVISION OF 151 MELANOBLASTOMATA ACCORDING TO THEIR ORIGIN IN CONGENITAL OR ACQUIRED LESIONS AND CORRELATED WITH SITE, AGE AND SEX INCIDENCE

		0–10		11–20		21–30		31–40		41–50		51–60		61–70		71–80		81–90		*Totals*	
		M	*F*	*M*	*F*	*M*	*F*	*M*	*F*	*M*	*F*	*M*	*F*	*M*	*F*	*M*	*F*	*M*	*F*	*M*	*F*
Head and Neck	C		1		2	3	1	3	2		1	3								9	7
	A					1	1					2	1	3	9	2	8	3	3	11	22
Upper Limb	C		1		1		3		5	2	1		1	1				1		4	12
	A						1	1	1			1	1	1						3	3
Lower Limb	C				5	6	4	5	9	1	7	1			1					12	26
	A						1		3		3	1	2			1	1			3	10
Trunk	C			1	1	2	1	2	2	5	5	2	1	2	1					14	11
	A								1	1		1					1			2	2

(A—Malignancy arising in an acquired lesion.)
(C—Malignancy arising in a congenital lesion.)

We do not attach any significance to the difference in numbers between the sexes, for if one goes further back into the records, the sex incidence becomes equal when the total reaches 500. A change in the recording system was introduced in 1950 and in this paper, for certain reasons of convenience, we have confined our investigations to the cases occurring since then.

DURATION OF PRE-EXISTENT MOLE

Many patients could not state the precise duration of their lesions prior to the appearance of features for which they sought treatment. In other cases the records are too vague to be of use: expressions such as "many years" mean different things at different age groups. However, there were 151 cases in which the lesion could, with reasonable certainty, be classified as having been present since childhood, or acquired in adult life. For the sake of convenience we have designated them "congenital" and "acquired." Congenital lesions are those which appeared during the individual's growth period and we have arbitrarily placed all lesions appearing before the age of 22 years in this category.

blastoma has usually exhibited its malignant nature by the age of 60 years. Acquired lesions of the head and neck were most frequently encountered in elderly women and in half of these we found that the total duration of a visible pigmented lesion had been less than three years. Subdivision of the lesions in the head and neck area has not contributed any further information.

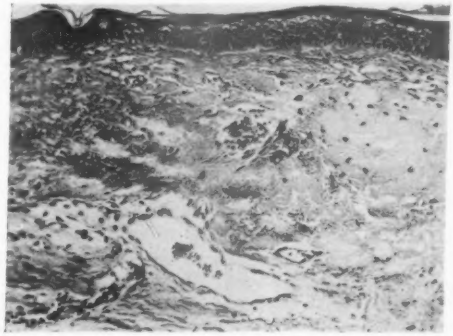

FIG. IV. Female aged 70 years; skin of face. Advanced collagen degeneration adjacent to a basal cell carcinoma. Note the resemblance to X-ray atrophy. Haematoxylin and eosin (x 150).

SOLAR EFFECTS

TABLE 3

CLASSIFICATION OF 151 MELANOBLASTOMATA ACCORDING TO SITE, PRESENCE OF COLLAGEN DEGENERATION AND ORIGIN IN CONGENITAL OR ACQUIRED LESIONS

		With C.D.		*Without C.D.*	
		M	*F*	*M*	*F*
Head and Neck	C	6	1	3	6
	A	9	20	2	2
Upper limb	C	1	1	3	10
	A	1	1	2	2
Lower limb	C	1		12	25
	A		1	3	10
Trunk	C			14	11
	A			2	2

(A—Malignancy in an acquired lesion.)
(C—Malignancy arising in a congenital lesion.)
(C.D.—Collagen degeneration.)

From Table 3 it is clear that cancer arising in acquired lesions of the head and neck was frequently associated with collagen degeneration. In most of these the collagen degeneration was severe and the epidermis had become atrophic. The type of lesion was that sometimes described as lentigo maligna or superficial melanoma. The usual form was a large flat junction lesion (Fig. V) in which a nodule of frankly invasive cancer eventually appeared (Figs. I, VI). Acquired lesions other than those of the head and neck seldom showed any histological effects of solar radiation in the dermis.

With melanoblastoma arising in a mole dating from childhood, collagen degeneration of any severity was seen in one case only. In a few others it was mild to moderate in degree. Early colloid degeneration was present in a few congenital lesions of the face and leg, indicating that penetration of the dermis by ultra-violet light had occurred, for this type of lesion is caused by ultra-violet light in the presence of subclinical deficiency of vitamin C. However, the degree of colloid degeneration which we encountered was so slight that we have not regarded it as having much significance.

To summarize, serious solar effects have been found in association with melanoblastoma mainly in acquired lesions of the head and neck of the elderly, particularly of elderly women.

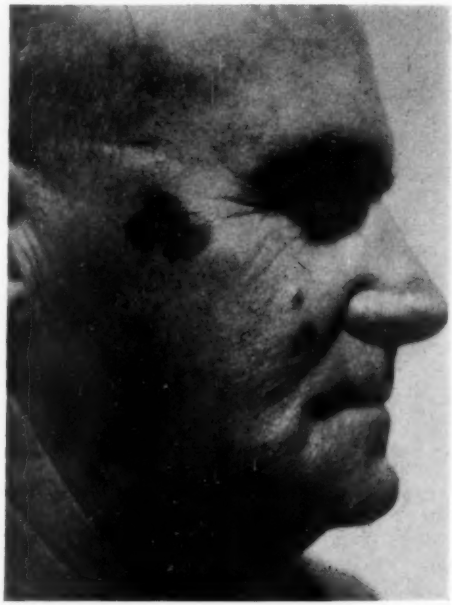

FIG. V. Male aged 65 years. Flat pigmented lesion which first appeared eleven years before. No evidence of malignancy. The patient refused excision.

OTHER FACTORS IN THE DEVELOPMENT OF MELANOBLASTOMA

(a) *Trauma*

In five patients there was a circumstantial history of trauma. In each case a pigmented lesion appeared at the site of trauma. One patient aged 57 years gave a history of having been "bitten on the leg by water bugs" eighteen months previously. When the rash subsided the skin of the leg had a crinkled appearance, and then, following sunburn twelve months later, a brown patch appeared. Over the next six months the edge of the lesion became raised. It was excised with the provisional diagnosis of pyogenic granuloma but histologically it had the typical appearance of melanoblastoma unaccompanied by collagen degeneration. Three other melanoblastomata related to trauma occurred at intervals of seven, fifteen and thirty years

after an injury which broke the skin. In the remaining case, the tumour occurred in the margin of an old burn.

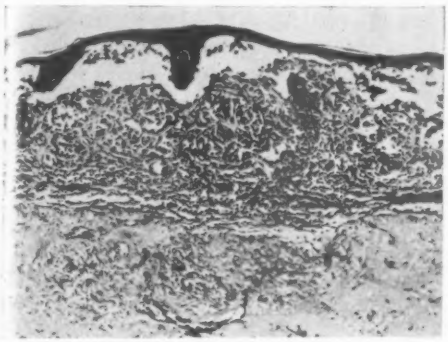

FIG. VI. Histology of the lesion illustrated in Fig. I, showing melanoblastoma and collagen degeneration of the dermis. Haematoxylin and eosin (x 150).

(b) *Pregnancy*

There were two cases in which life-long moles became malignant during pregnancy. One was situated on the back of a woman aged 30 years and the other was on the ankle of a patient aged 35 years. The second patient developed another primary growth a year later. In a third patient malignancy occurred during pregnancy in a pigmented spot on the leg but it is not certain whether the original lesion was congenital or recently acquired.

(c) *Multiple primary growths*

Six patients exhibited more than one primary growth. These were recognizable by the origin of the tumour from proliferated melanoblasts at the junction of dermis and epidermis, a feature which distinguishes them from secondary melanotic implantations.

(1) Female child aged 6 years with xeroderma pigmentosum. This child developed a melanoblastoma of the cheek and a few months later another primary growth appeared on the arm. She has since succumbed to metastases. There were no histological changes attributable to solar radiation.

(2) A young woman aged 18 years who developed malignancy in a compound naevus of the face. Four years later a pigmented lesion appeared on the leg and in a few months became malignant. Histological preparations of the facial lesion shows very early colloid degeneration in the vicinity but no collagen changes.

(3) A male aged 37 years in whom malignancy occurred in a long standing mole of the arm. Shortly afterwards the attending surgeon noticed a further melanoblastoma on the back of the neck, of which the patient was unaware. There was no histological evidence of solar effects.

(4) A male aged 51 years had a melanoblastoma removed from his shoulder together with axillary metastases. Six months later, a new primary growth appeared on the face. There had been no previous mole. It is not known whether the first growth arose in a pre-existent mole or not.

(5) An elderly woman aged 74 years developed 3 separate growths of the face over a period of one year. Four years later she developed another primary growth. She had very severe collagen degeneration of the skin with areas of atrophic epithelium in which the melanoblastomata arose.

(6) Female aged 35 years had a mole on her ankle which commenced to grow during pregnancy. It was excised and the inguinal lymph nodes were removed. A year later a new primary melanoblastoma arose on her leg.

DISCUSSION

Pack (1954) believes that all moles are probably congenital and that they often exist in microscopic form until the stimulus of puberty causes them to enlarge and become apparent. Nevertheless, there is a group in our series in which the first appearance of a mole has been in the later years of life. Most of these patients have exhibited marked collagen degeneration of the skin and have presented with a typical lesion, consisting of a flat irregularly pigmented area of the head or neck, with a nodule of invasive tumour arising in it. Certainly, in a few cases melanoblastoma has occurred in the absence of collagen degeneration, but this does not invalidate our contention that the presence of collagen degeneration predisposes to the development of melanoblastoma. Other carcinogenic influences acting over long periods

may play a part also and may be responsible for the cases unaccompanied by collagen degeneration.

The high incidence of collagen degeneration in this group of acquired lesions of the head and neck seems more than fortuitous. With age there is a rising incidence of melanoblastoma of the head and neck. There is also a rising incidence of collagen degeneration because of the cumulative effect of ultra-violet light on the dermis.

That collagen degeneration affects the metabolism of melanocytes in the basal region of the epidermis is illustrated by the mottled and irregularly pigmented appearance in uncomplicated collagen degeneration even though the epidermis itself is atrophic. At the time of development of the collagen degeneration there must have been insufficient pigment in the basal cells to prevent the penetration of ultra-violet light. When the metabolism of the skin has been profoundly altered by severe collagen degeneration of the dermis irregular pigmentation appears. Hyperplasia of melanocytes is a step further and once a flat mole with junctional activity has been established, malignancy can be expected to follow. We believe that severe collagen degeneration of the dermis predisposes to the development of melanoma as well as to keratoses, squamous cell carcinoma, keratoacanthoma and basal cell carcinoma (Mackie and McGovern, 1958).

Malignancy supervening upon congenital moles was most common in the lower limbs of females. This, too, suggests that exposure may have played a part although only a few minor effects of solar radiation were demonstrable. Possibly an inherent tendency was accelerated by exposure to sunlight.

Factors which cause the persistence into adult life of junctional activity in congenital moles, and factors which then precipitate malignancy in them, are not known with any certainty, though both hormonal influences and trauma seem to play some part.

SUMMARY

Pigmented moles acquired in adult life which subsequently became malignant appeared chiefly in the head and neck of elderly people in whom there was pronounced alteration in structure of the dermis as a result of solar radiation. We were unable to demonstrate significant degrees of collagen degeneration of solar origin in any other group of melanoblastoma patients. These facts suggest that the carcinogenic action of ultra-violet light is mediated through its action on the dermis.

ACKNOWLEDGEMENTS

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STUDIES ON RENAL TRANSPLANTATION IN SHEEP

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FOLLOWING early experiments on transplantation of the kidney by Carrel (1902), a large body of literature on this subject has accumulated. Several articles are available in which the history of renal grafting has been reviewed (Dempster, 1950; Hume *et alii*, 1955). An outstanding contribution to our knowledge of the behaviour of renal transplants came from Carrel (1906, 1911) who described the survival of a dog for two and one-half years after double nephrectomy with re-implantation of one kidney. Williamson (1923) showed that whereas a re-implanted or autografted kidney functioned well, a kidney from another animal or homograft, after functioning for four to ten days became oedematous and anuric. He recognized that biological rather than technical factors limited the function of such homografted kidneys. Later Williamson (1926) described clearly the histological changes associated with homograft rejection. The next major advance came from the work of Simonsen *et alii* (1953) who showed that a second homografted kidney from the same donor was destroyed at a faster rate than the first. Previous transplants of spleen or skin produced this same accelerated destruction of a kidney homograft from the same donor, demonstrating that an acquired tissue immunity was involved in renal homograft destruction. Dempster (1953b) showed that not only does the host react violently to a foreign kidney but also the kidney itself reacts against its host thereby accelerating its own destruction.

Many unsuccessful attempts to perform renal homografts in man have been described. In some, surprising results have been achieved with function up to five months (Hume *et alii*, 1955) but present knowledge suggests that all are doomed to failure with one exception. In identical twins it has been shown that a renal homograft will function satisfactorily (Merrill *et alii*, 1956; Murray *et alii*, 1958) and results indicate that the "take" appears to be permanent, at least for the period of follow-up which is now approximately three years.

Recent work on the induction of tolerance to homografts (Billingham *et alii*, 1956a, b) gives promise that we may in the future be able to break down the biological barriers to the homografting of tissues. This possibility makes careful evaluation of the functional establishment of renal grafts, of increasing interest and it was with this in mind that the behaviour of transplanted kidneys in sheep has been investigated in the present study.

MATERIALS AND METHODS

Experiments were carried out on adult Romney Marsh sheep under general anaesthesia using aseptic precautions. All kidneys were transplanted into the iliac fossa but the precise technique varied in autografts and homografts. The left kidney was used for grafting since in the sheep a longer renal pedicle is available on this side. The renal vessels were divided close to the aorta and vena cava and the ureter sectioned 3 inches below the renal pelvis. The dissection of perinephric fat from the hilum and renal vessels was made easier by keeping the renal vein distended with blood. Several venous tributaries required ligation and once the renal vein collapsed it was difficult to identify these. When cleared of fat the kidney was emptied of blood and the vessels rinsed with heparo-saline (1 mg./ml.), taking care not to allow this to enter the kidney. Iliac grafts were used anastomosing the divided external iliac artery to the renal artery. The renal vein was sutured end to side to the inferior vena cava using Blalock's technique with a Satinsky clamp to control blood flow. In autografts ureteric anastomosis was used but in homografts cutaneous ureterostomy was necessary. Heparin was given for the first eight hours post-operatively and penicillin (500,000 units/day) was used for the first four days. The animals were kept in pens,

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being fed on hay and prepared mash with freely available water, but in long-term survivals the animals were transferred to the open field. Urinary collections were made using a perspex box strapped to the abdomen in male animals (Fig. I). In female animals a urethral catheter was stitched into the bladder, while the output from the grafts was collected by means of a polythene catheter attached to a polythene bottle fixed to a leather harness. The specific gravity, urea, protein and electrolyte content of normal urine and graft urine were measured. Periodic blood samples were taken for non-protein nitrogen and electrolyte estimations. Following cessation of function, grafts were excised, and specimens taken for histological examination. Immediate fixation in Helley's fluid was carried out and 5μ sections were stained by haematoxylin and eosin, Van Gieson, a modified Masson's trichrome and PAS (Lillie, 1951). Plasma cells were stained by Unna's method (Gurr, 1952). Microdissections were carried out on autografted kidneys but proved impossible on homografts due to extreme friability of the nephrons.

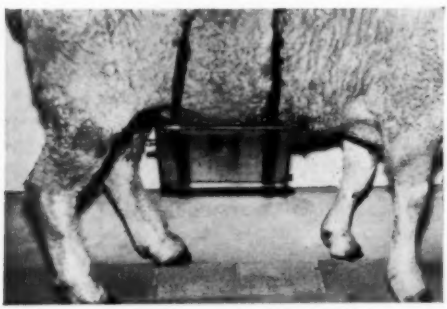

FIG. I. Urine collection box in position. In male animals the box fits over the penis. The box is triangular in cross section to allow the animal to lie down in comfort.

OBSERVATIONS

1. *Renal autotransplants*

In order to study the long-term function of autografts it was considered essential to perform abdominal transplantation and to re-establish normal urinary flow. A series of 9 female animals were used. With the animal lying on its back a catheterizing cystoscope was passed and the left ureter catheterized with a No. 3 French catheter. The abdomen was now opened through a long left paramedian incision Fig. II) and the left external iliac artery and vena cava prepared for anastomosis. The left kidney was now excised, cleared of fat and then re-implanted intraperitoneally into the left iliac fossa (Fig. III). Uretero-ureteric anastomosis was considered necessary since insufficient vascularized length of ureter was available to reach the bladder. The ureter was sutured over a polythene tube (1 mm. bore) which led from the renal pelvis across the anastomosis through the bladder and urethra to the exterior. Four fine silk sutures were used across the outer layers of the ureter to effect the anastomosis. The polythene splint was introduced from above downwards using the indwelling ureteric catheter as a leader. This technique allowed collection of urine from the graft and also avoided leakage of urine and subsequent stricture of the suture line. The opposite normal kidney was removed two weeks after autotransplantation of the left kidney.

Six of these grafts were considered technically successful, secreting satisfactory volumes of urine (Table 1). Three long-term successes were obtained, the animals surviving in good health for six months, twenty-four months and thirty months, when they were killed. In these kidneys the period of occlusion of circulation during the transplantation procedure varied from one hour eight minutes to one hour twenty minutes. It was customary for the ureter to begin to discharge urine approximately twenty minutes after the release of circulation through the graft, this urine being heavily contaminated with blood. Analysis of the urine production by these grafts showed suppression of urine for some days following transplantation, with progressive recovery over a period of several weeks. Urinary specific gravity was usually in the region of 1020 for the first few weeks. Urinary sodium and potassium levels approximated to normal and urea content varied from 1.3 to 2.9 G/100 ml. Biopsies carried out on the transplants at one week showed a normal histology. Indigo-carmine excretion carried out one week after operation showed no secretion from the graft but following removal of the opposite normal kidney one week later good concentration of dye appeared in two and one-half minutes. Intravenous pyelography carried out in one animal

only, at seven days, showed no function in the transplant. This investigation was not satisfactory in sheep owing to the small size of the renal pelvis. All three successful animals continued to thrive and grow satisfactorily. An examination of urinary output up to two and one-half years post-operatively showed normal urinary volumes, with specific gravity approximately 1030. Urea concentration varied from 2 to 4 G/100 ml. The sodium and potassium content of the urine was normal with no protein leakage. Periodic increase in intrapelvic fat (Fig. IV), but otherwise the appearances of the kidneys in the surviving animals were normal. Arterial and venous anastomoses were almost invisible, while the ureteric anastomosis could be seen as a thin ridge on the surface of the ureter. Histological examination of these kidneys showed hypertrophy of renal tubules and glomeruli with slight increase in interstitial fibrous tissue (Fig. V). Microdissection showed the individual nephrons to be normal anatomically.

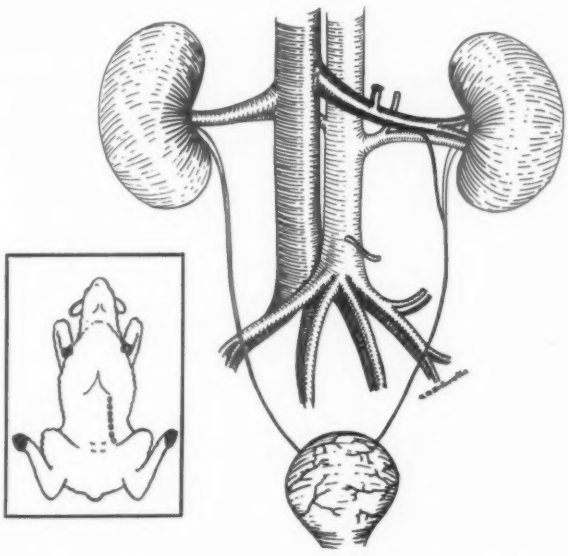

FIG. II. A drawing of the great vessels and urinary system of the sheep. Insert shows the incision for the intraperitoneal autograft procedure.

samples of blood from these three animals showed normal serum and potassium levels and the blood non-protein nitrogen remained steady after secondary nephrectomy, except in the case of one animal where it rose to 63 mg./100 ml. one week after operation. At the time of killing the T.N.P.N. was 65, 44 and 48 mg./100 ml. for the animals killed at six, twenty-four and thirty months respectively. The grafted kidneys at autopsy had all drifted from the iliac fossa and were found surrounded by dense fibrous tissue and adherent to loops of adjacent intestine. Macroscopic appearances of these autografts showed some atrophy of the cortex and slight

In three further animals the graft appeared to be satisfactorily established but when the opposite kidney was removed graft function proved to be inadequate. The animals became extremely emaciated, developed an intractable diarrhoea, and became caste, with typical Kussmaul breathing and urea frosting of the nose. Progressive increase in blood non-protein nitrogen accompanied these changes and the animals died in uraemia eleven, twelve and twenty-one days after the right nephrectomy. In these cases the period of ischaemia had exceeded one and one-half hours. Examination of these grafts showed severe tubular necrosis with patchy areas of

leucocytic infiltration and in one case the kidney had been completely destroyed and converted into necrotic liquid material. Three technical failures occurred due to renal vein thrombosis with venous engorgement of the kidney, renal artery thrombosis with infarction, and in the third animal damage to the ureter during the clearance of perinephric fat gave rise to a traumatic hydronephrosis. Leakage of urine caused a cystic collection around the graft necessitating its removal.

laterally nephrectomized sheep (Table 2). After periods of ischaemia varying from two to three hours the circulation to the iliac graft was restored. Three kidneys were cooled by arterial perfusion and in two of these satisfactory function was obtained. These two animals were killed after six days and twenty-one days, at which times their blood non-protein nitrogen was 57 and 66 mg./100 ml. respectively. Examination of the grafts showed a surprising degree of renal infarc-

TABLE 1

LEFT RENAL AUTOGRAFTS WITH CONTRALATERAL NEPHRECTOMY

Sheep number	*Period of ischaemia*	*Graft function*	*Histological appearances*
105	1 hr. 8 mins.	Oliguria 1 week then 100–300 ml./day. Animal killed 6 months (N.P.N. 65)	Glomeruli and tubules hypertrophied. Some interstitial fibrosis
8	1 hr. 10 mins.	Oliguria 1 week then 300–400 ml./day. Animal killed 30 mths. (N.P.N. 48)	Glomeruli and tubules hypertrophied, otherwise normal.
82	1 hr. 20 mins.	Initial oliguria then 400–500 ml./day. Animal killed 24 months. (N.P.N. 44)	Hypertrophic changes, otherwise normal.
13	1 hr. 40 mins.	Oliguria. Never established satisfactory function. Died uraemia 11 days. (N.P.N. 440)	Not possible—graft necrotic.
104	1 hr. 55 mins.	Oliguria. Died uraemia 12 days. (N.P.N. 320)	Extensive tubular necrosis—many tubules converted into eosinophilic debris. Polymorph infiltration.
81	2 hrs.	Oliguria. Died uraemia 21 days. (N.P.N. 360)	Tubular necrosis with patchy areas of fibrosis. Glomeruli relatively normal.

Renal cooling

It has been shown that periods of renal ischaemia longer than one hour are uniformly fatal in unilaterally nephrectomized sheep (Mitchell and Woodruff, 1957). Since abdominal grafting commonly necessitated occlusion of the renal circulation for longer periods than this it was considered important to investigate the effects of local renal cooling in preventing tubular damage. Two methods of producing local renal cooling were studied. Firstly, intravascular cooling of the kidney by perfusion with cold dextran (6 per cent. in normal saline), and secondly surface cooling by invagination of the kidney into a plastic bag through which cold saline was circulated. A needle thermocouple inserted deeply into the kidney allowed continuous recording of the intrarenal temperature. Renal autografts cooled by perfusion with dextran at 0°C were performed in 5 uni-

tion (Fig. VII). The third graft was anuric and when explored twenty-four hours after operation, thrombosis of the renal artery was found. Two further autografts were made using retrograde perfusion via the renal vein at pressures of 20 mm. and 30 mm. of mercury. In both cases tubulovenous rupture occurred with rapid flow of dextran from the ureter. Neither of these two grafts established satisfactory urine production and when explored six days later both were completely necrotic. It was concluded from these experiments that arterial and venous perfusion using dextran was an unsatisfactory method of renal cooling.

Surface cooling of the kidney to between 10° and 20°C by saline immersion using a plastic bag to prevent saline contact has been shown to produce a striking increase of tolerance to ischaemia without demonstrable

ll effects from hypothermia (Mitchell and Woodruff, 1957). The remaining transplants described in this paper were therefore cooled by this technique.

incised and the external iliac artery and inferior vena cava exposed extraperitoneally. The left kidney from a simultaneously anaesthetized animal was now excised and cleared

TABLE 2

AUTOGRAFTS COOLED BY DEXTRAN PERFUSION

Sheep number	*Period of ischaemia*	*Graft function*	*Histological appearances of graft.*
20	1 hr. 55 mins. (Arterial perfusion)	Satisfactory function. Killed 6 days	Tubular necrosis—some polymorph infiltration
155	2 hrs. 55 mins. (Arterial perfusion)	Satisfactory function. Killed 21 days	Areas of infarction with heavy polymorph infiltration
107	3 hours (Arterial perfusion)	No function. Graft removed 24 hrs.	Renal infarction
209	1 hr. 30 mins. (Venous perfusion)	No function—animal died 3 days	Graft liquefied
206	1 hr. 50 mins. (Venous perfusion)	No function—explored 6 days	Graft liquefied

2. *Renal homografts*

Twelve male sheep were used as recipients in homograft experiments. With the animal in the left lateral position the right loin was

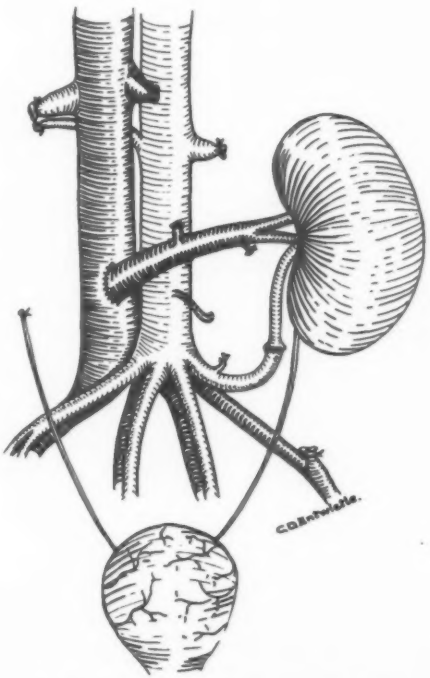

FIG. III. Iliac autograft in position. The normal right kidney has been removed.

of fat. It was placed in a plastic bag and cooled in saline at 10°C while the donor incision was sutured. The kidney was now reimplanted extraperitoneally into the recipient animal which now had three functioning kidneys. The ureter was led out through a separate stab wound and cannulated with a polythene tube which was led to a polythene bottle strapped to the sheep's side.

The results of these experiments are summarized in Table 3. In five of the animals after an initial period of oliguria lasting twenty-four hours, the transplant functioned well for two to three days. Progressive reduction in urinary volume occurred, followed by anuria on the fifth day. Specific gravity of graft urine tended to be fixed (1010 to 1020), and the urea content low (1 to 1.5 G/100 ml.). There was some slight protein leakage from homografted kidneys. Sodium conservation by the graft was not as good as that of the normal kidneys but potassium level in the graft urine appeared to be normal. Blood non-protein nitrogen was measured at two-day intervals and remained normal while the graft was functioning. In one of the grafts good volumes of urine were collected for nine days before anuria developed (Fig. VIII). In one animal only, both normal kidneys were excised at the time of grafting and satisfactory volumes of urine continued for twenty-eight days before anuria occurred (Fig. IX). Three technical failures occurred, two due to renal artery thrombosis in the early post-operative period while in

the third inadvertent kinking of the renal vein resulted in the graft splitting when the arterial clamps were released. The grafts were removed from the recipient animals at the onset of anuria, or soon afterwards. The kidneys were twice their previous size and showed swelling and oedema, particularly of the renal cortex. The ureter was also grossly oedematous. The graft was surrounded by sero-sanguinous fluid and there was no evidence of adhesion between the graft and the tissues of the donor animal. If removal of the graft was delayed twenty-four to forty-eight hours after the onset of anuria complete

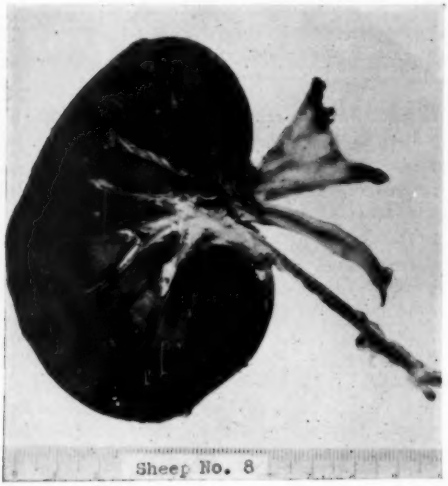

FIG. IV. Photograph of the "iliac" autograft from sheep No. 8. This kidney was ischaemic one hour ten minutes during transplantation. It maintained the animal in good health for two and one-half years when the sheep was killed. Blood T.N.P.N. at this time 48 mg./100 ml.

liquefaction due to autolysis occurred. Histological examination of the homografts showed separation of the tubules by oedema with round cell infiltration (Fig. X). The tubules and glomeruli appeared relatively normal apart from heavy infiltration of glomerular tufts with round cells. The glomerular capillaries were empty. The peritubular capillaries, however, were congested, particularly in the juxtamedullary area. Mononuclear cells were concentrated around the individual glomeruli and also around peritubular capillaries suggesting a possible vascular orgin for these cells. Mononuclear cells appeared first in focal areas but increased until at a later stage the interstitial tissue was stuffed with round cells. The predominant cellular pattern consisted of plasma cells and lymphocytes but few polymorphonuclear leucocytes were seen. Urine cultures and cultures from renal

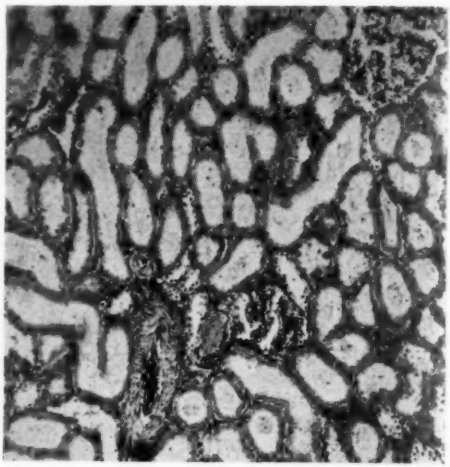

FIG. V. Photomicrograph of autograft from sheep No. 8. This shows tubular hypertrophy. The glomeruli are large but normal and no interstitial changes are seen. Stain haematoxylin and eosin. (x 300)

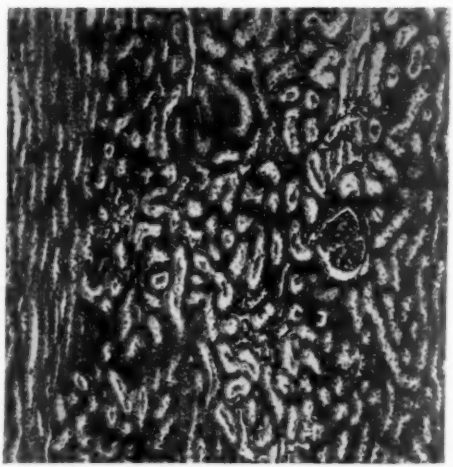

FIG. VI. Photomicrograph of a normal sheep kidney. Compare with Fig. V. Stain haematoxylin and eosin. (x 300)

tissue were negative in the majority of homografts so that these pathological appearances were not due to infection.

FIG. VII. Photograph of the iliac autograft from sheep No. 155. This kidney was rendered ischaemic for two hours fifty-five minutes after intravascular cooling by perfusion with dextran solution at 0°C. Maintained the animal for three weeks when it was killed. Blood N.P.N. at this time was 66 mg./100 ml. Note the extensive infarction of the cortex.

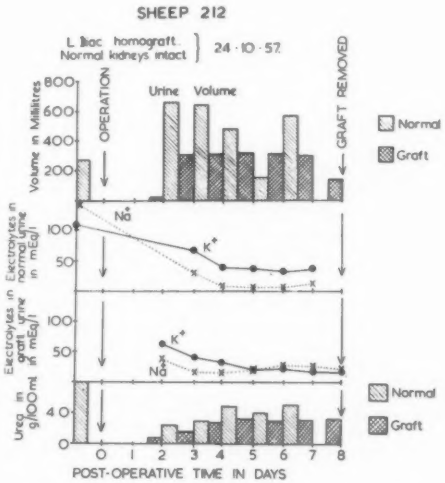

FIG. VIII. A chart to show the difference between normal urine and graft urine in sheep No. 212 with three functioning kidneys. The volume of the graft urine in this case was higher than usual. Urea concentration is lower in the graft urine than in normal urine.

Renal function in recipient animals

An unusual feature of the recipient animals after removal of the homograft was a considerable weight loss with frequent slow healing of the re-opened loin incision. A large number of wound infections occurred in these animals, whereas in autografts or other renal operations in sheep, wound infection was uncommon. In several animals blood non-protein nitrogen rose progressively after removal of the graft and one animal died in uraemia. This suggested the possibility that the homograft had produced immunological injury to the recipient's own kidneys. This problem was therefore studied in a further three animals, where urine collections and blood samples were continued for some time after the homograft had been removed. The right kidney was removed for histological study when the anuric homograft was excised, leaving the animal dependent on its remaining left kidney. While the homograft was functioning, the animal's own kidneys produced smaller volumes of urine than normal. After its removal and simultaneous right nephrectomy, no striking alteration in kidney function was observed, apart from occasional proteinuria. Blood non-protein nitrogen did not change in these animals. In two animals of this group, where excision of the anuric homograft had been delayed for forty-eight hours, cultures from both the homograft and the normal right kidney grew B. coli and Cl. welchii. The right kidney in these animals was soft with subcapsular haematoma forma-

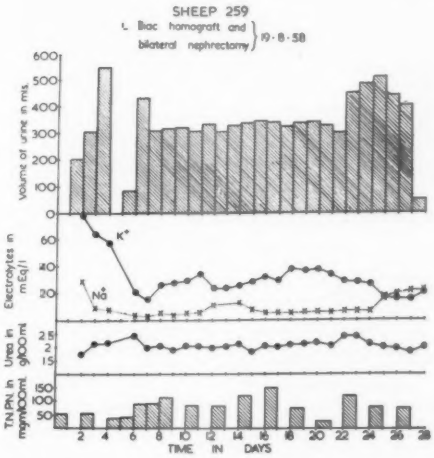

FIG. IX. A chart showing urine collections from sheep No. 259 existing solely on a homograft. Function is prolonged and considerably greater volumes of urine are produced.

tion, and histologically revealed moderate tubular necrosis but no interstitial cellular changes. Presumably these changes were due to Cl. welchii toxaemia as were the more severe tubular changes which resulted in renal failure in the single animal dying in uraemia after homograft removal. With the onset of anuria in a homograft, renal circulation became obstructed and these organisms were established in the autolysing graft if it was not removed within twenty-four hours. The alimentary canal was the likely source of these organisms.

coitum laparotomy was carried out and the embryos injected through the uterine wall with 0.5 to 1 ml. of spleen cell suspension (25 to 50,000 cells/cu. mm.). Five live lambs were obtained and at six months of age each received a renal homograft from the original donor of spleen cells. Both kidneys were left intact in these lambs. Two of these renal grafts were unsuccessful due to renal artery thrombosis while a third was anuric but viable when removed one week after operation. The remaining two grafts passed satisfactory urine volumes at first but anuria

TABLE 3

RENAL HOMOGRAFTS — BOTH KIDNEYS OF RECIPIENT PRESENT

Sheep number	*Period of ischaemia*	*Graft function*	*Histological appearances of graft*	*Histological appearance animal's own kidneys*
167	20 mins.	Secreted for 4 days with complete anuria 5th day—removed.	Marked oedema with tubules widely separated. Heavy interstitial round cell infiltration.	—
213	1 hr. 35 mins.	Secreted—30–40 ml./day with complete anuria 6th day—removed 24 hrs. later.	Marked oedema. Glomerular capillaries collapsed. Heavy round cell infiltration around tubules and glomeruli.	Right kidney. Normal.
214	1 hr. 35 mins.	Secreted approximately 20 ml./day for 4 days. Anuria 5 days—removed.	Typical appearances. Glomerular capillaries empty. Peritubular oedema and mononuclear infiltration.	Right kidney. Some casts in the collecting tubules. Otherwise normal.
217	1 hr. 15 mins.	Secreted approximately 40 ml./day for 3–4 days. Progressive reduction. Anuric on 6th day—removed.	Very marked interstitial round cell infiltration. Glomerular capillaries empty.	Right kidney. Normal.
198	1 hr. 20 mins.	Oliguria 5 days with anuria on 6th day—removed.	Glomeruli swollen—capillaries empty. Numerous granular casts. Tubules dilated with peritubular oedema. Considerable interstitial round cell infiltration.	—
212	1 hr. 10 mins.	Oliguria 48 hours then 300 ml./day. Anuria on 9th day—removed.	Heavy interstitial round cell infiltration. Some tubular necrosis. Many tubules dilated. Occasional amorphous casts. Glomerular capillaries empty.	Right kidney (2 weeksT.N.P.N. 250). Marked degree of Tubular necrosis. Numerous casts. Glomeruli normal.
173	1 hr. 45 mins.	Oliguria 48 hours. Anuria 2 days—removed.	No interstitial reaction but complete glomerular and tubular necrosis. Tubules full of epithelial debris (renal artery thrombosis).	—
208	1 hr. 10 mins.	Kidney failed to secrete urine after 24 hours—removed 48 hours.	Glomerular capillaries normal. Some interstitial round cell infiltration in focal areas. Occasional casts. No peritubular oedema.	

Attempts to induce tolerance to renal homografts

It has been shown (Billingham *et alii*, 1956a) that injection of foreign spleen cells into foetal mice induces tolerance to subsequent skin grafts from the same donor. It was considered important to test this observation in relation to kidney homotransplants. A series of pregnant ewes of known conception dates was used. At sixty to ninety days post-

occurred at six days and thirteen days respectively. When removed at this time these kidneys were typically oedematous and histological examination showed the picture of round cell infiltration similar to the homografted kidney from uninjected recipient animals. The kidney which failed to produce urine after grafting showed similar changes. The intra-embryonic injection of spleen cells, as evidenced by the two successful homo-

grafts, did not appear to influence their rate of destruction or the immunologically induced cellular reaction.

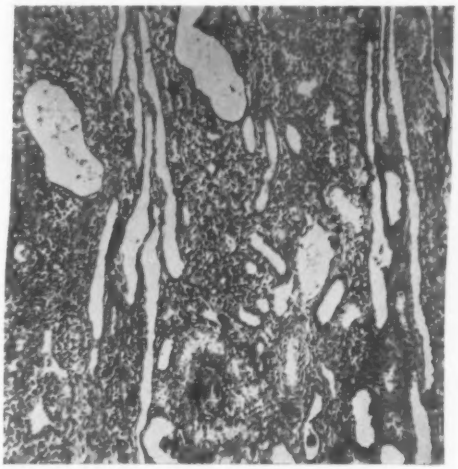

FIG. X. Photomicrograph of renal homograft from sheep No. 212. Removed after onset of anuria nine days after operation. There is marked interstitial infiltration with round cells and some hydronephrotic changes due to the indwelling ureterostomy tube. Many of the tubules are necrotic. Stain haematoxylin and eosin. (x 300)

about 1010. Although they respond to water diuresis, inability to concentrate remains a permanent feature (Dempster, 1950).

FIG. XI. Photomicrograph of renal homograft from sheep No. 477. The lamb had received an injection of spleen cells from the donor of this kidney when in utero. Similar histological changes are seen as occur in homograft rejection, cf. Fig. X. Stain haematoxylin and eosin. (x 300)

DISCUSSION

The results reported here on iliac autografts confirm that such kidneys are capable of maintaining an animal in perfect health for up to two and one-half years. Two previous studies of long-term function in abdominal autografts (Dempster *et alii*, 1955; Murray *et alii*, 1956) have revealed similar findings. In order to attain satisfactory long-term function, however it is essential that the ureter is connected to the bladder rather than to the skin surface by ureterostomy. Dempster (1950) has shown that ureterostomy in experimental animals is not satisfactory for long-term study due to obstruction and ascending infection. The urea concentration and specific gravity of the long-term autografts in this paper were normal, indicating that when urinary flow is reconstituted and the intra-abdominal site is chosen, an autograft behaves exactly like a normal kidney. Cervical autografts, however, exhibit a permanent polyuria with specific gravity fixed at

The results in homografted kidneys confirm the findings of other workers that such grafts are destroyed in a short time as a result of an immunological reaction. It is interesting that the function of such a homograft is profoundly affected by the presence of the animal's own kidneys. In animals which have three kidneys the graft produces only small volumes of urine (Table 3). If on the other hand bilateral nephrectomy is carried out at the time of grafting, urinary output is much greater (Fig. IX). This emphasizes the importance of renal loading, a concept brought forward by Hinman (1923) in relation to his theory of renal counterbalance in hydronephrosis. The renal load in the autograft experiments reported here, was also important since satisfactory function from the graft was not established until the opposite normal kidney had been removed. The length of time a homograft functions also appears to be influenced by the presence of the animal's normal kidneys. When both normal kidneys are present, anuria in the

homograft occurs about the fifth day but if the homograft is the sole kidney it may function up to twenty-eight days (Fig. IX). An increased survival of homografts in goats after bilateral nephrectomy was recorded by Humphries (1956), although he attributed this to a generic difference in time of rejection. It was interesting in the present study that over the period when the animals were carrying three kidneys in no case did the blood non-protein nitrogen fall below normal levels, although on a theoretical basis this might be expected to occur (Platt, 1952).

It is conceivable that a homografted kidney could produce immunological damage to the host's own kidneys. The fact that the host develops a toxic syndrome at the time that the homograft becomes anuric has been recorded by Dempster (1953a). Ishikawa *et alii* (1957) have referred to a hepato-renal disturbance as a result of a renal homograft which produced lethargy, anorexia, pyrexia and rapid loss of weight in the recipient. Histologically they described round cell infiltration of the recipient's normal kidneys resembling that in the homograft and suggested that the transplant and recipient kidneys acted as antigens to each other. In the present study histological examination of recipient kidneys failed to reveal any round cell infiltration and bacteriological study of the homograft and normal kidneys suggested that in those animals where renal damage occurred in the recipient kidneys, Cl. welchii toxaemia was the probable explanation.

The fact that embryonic spleen cell injections failed to induce tolerance to kidney homografts is not surprising. Such methods could only succeed in modifying the host against graft reaction without affecting the graft against host response. In successful homotransplantation of a whole organ, which must be regarded as unequal parabiosis, both aspects of this immunological process require modification before permanent survival can be attained.

SUMMARY

Transplants of the kidney were performed in 34 sheep using the "iliac" position. The function of autografts, homografts and normal host kidneys was studied together with the histological changes. To reduce ischaemic damage during transplantation, the kidneys were cooled. Cooling by perfusion was unsuccessful but a useful technique of local renal cooling was evolved. Iliac autografts functioned normally up to two and one-half years but homografts were rejected in five to nine days. The function of a grafted kidney was affected by the presence of normal kidneys. The concept of renal loading in relation to graft function is discussed. Bacteriological study showed that an anuric homograft, if not quickly removed, became secondarily infected with Cl. welchii. This led to toxic damage to the animal's own kidneys. Injection of spleen cells into embryonic lambs was followed six months after birth by kidney homotransplantation from the same donor. There was no increased tolerance to such renal homografts.

ACKNOWLEDGEMENTS

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THE GENETIC APPROACH TO HEREDITARY CONGENITAL PTOSIS

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THE hereditary nature of many of the congenital malformations encountered by the surgeon is readily observed, but the contribution which a knowledge of the genetics of the condition may make to an understanding of the origin and form of the defect is often overlooked. It is the purpose of this paper to show in what way information which at the present time is usually wasted can be utilized by the geneticist, and how apparently commonplace data are of value if properly recorded.

Familial ptosis of a non-congenital type in which the age of onset is usually 40-50 years is now well authenticated (Rodin and Barkan, 1935; Faulkner, 1939; Hamilton, 1951). The present study deals only with hereditary congenital ptosis, which both genetically and in its natural history is unrelated to the non-congenital defect.

Identification of the gene or gene complex associated with an hereditary trait does not depend entirely on an identical manifestation of this particular trait in all affected individuals. Each gene is characterized by specific linkage and dominance relations, by its interactions with other genes, and by its time of action during development. These attributes may be modified by environmental or genetic agencies, so that the ultimate effect of any particular gene is determined by an overall genetic constitution, and the environment of the individual.

Classification of Ptosis Cases

Clinically three main types of hereditary congenital ptosis may be recognized:

1. Simple ptosis.
2. Ptosis associated with epicanthus.
3. Ptosis associated with external ophthalmoplegia.

1. Simple ptosis (Fig. I), which may be either unilateral or bilateral, is almost invariably due to a failure in the ultimate differentiation of the extrinsic muscles of the eye, and the superior rectus may be deficient as well as its derivative, the levator palpebrae. Spaeth (1943) found that simple ptosis represented about 77 per cent. of all cases of congenital ptosis.

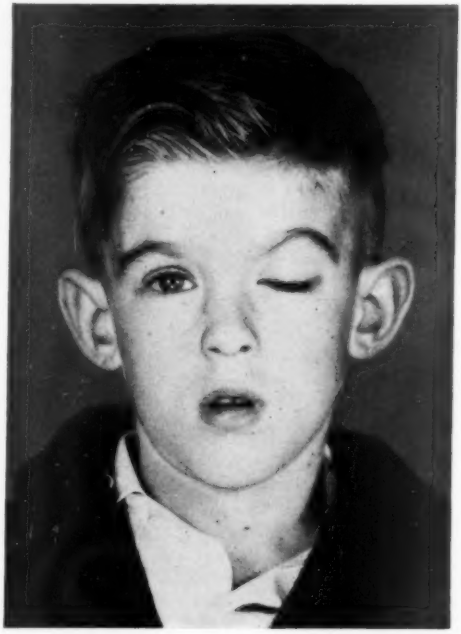

FIG. I. Simple unilateral ptosis.

2. In the case of ptosis associated with epicanthus (Fig. II) the ptosis is again caused by the absence or impairment of function of the levator palpebrae. The condition is usually bilateral and symmetrical.

Rodin (1936) and other authors have considered blepharophimosis (Fig. III), in which the size of the palpebral aperture is greatly reduced, to be a separate condition. The distinction appears to be merely one of degree.

FIG. II. Ptosis with epicanthus.

3. In ptosis with external ophthalmoplegia, a much less common condition, there is a more widespread, though seldom complete, paralysis of the extra-ocular muscles. Limited investigations indicate that external ophthalmoplegia is due to agenesis or aplasia of the oculomotor nucleus (Duke-Elder, 1949) rather than any failure of muscle differentiation.

Waardenburg (1924) described a family in which a recessive gene is apparently responsible for this defect, but the majority of published pedigrees suggest that a dominant autosomal (not sex-linked) gene is more frequently involved.

The discussion which follows will be restricted to the more common types of ptosis with or without epicanthus.

PEDIGREES OF PTOSIS

Owing to the small size of human families, a certain number of sibships in which affected individuals are expected to occur fail to show evidence of the particular trait, while others will contain more than the expected proportion of defectives. The tendency to collect for publication only those more spectacular pedigrees which show a high incidence of the defect introduces a serious bias into the statistics on which the geneticist depends. Every effort should therefore be made to investigate all pedigrees showing the inheritance of rare conditions such as congenital ptosis, for only in that way will accurate studies become possible.

FIG. III. Ptosis with severe epicanthus, "blepharophimosis."

Six pedigrees of congenital ptosis, five from Tasmania and one from Victoria, are shown in Fig. IV. Contact was made with the propositi when they attended for consultation or operation at a plastic surgery clinic, and in each case the family history was thoroughly investigated. There is no evidence of consanguineous marriage in any of the pedigrees, and inheritance is through one side of the families only.

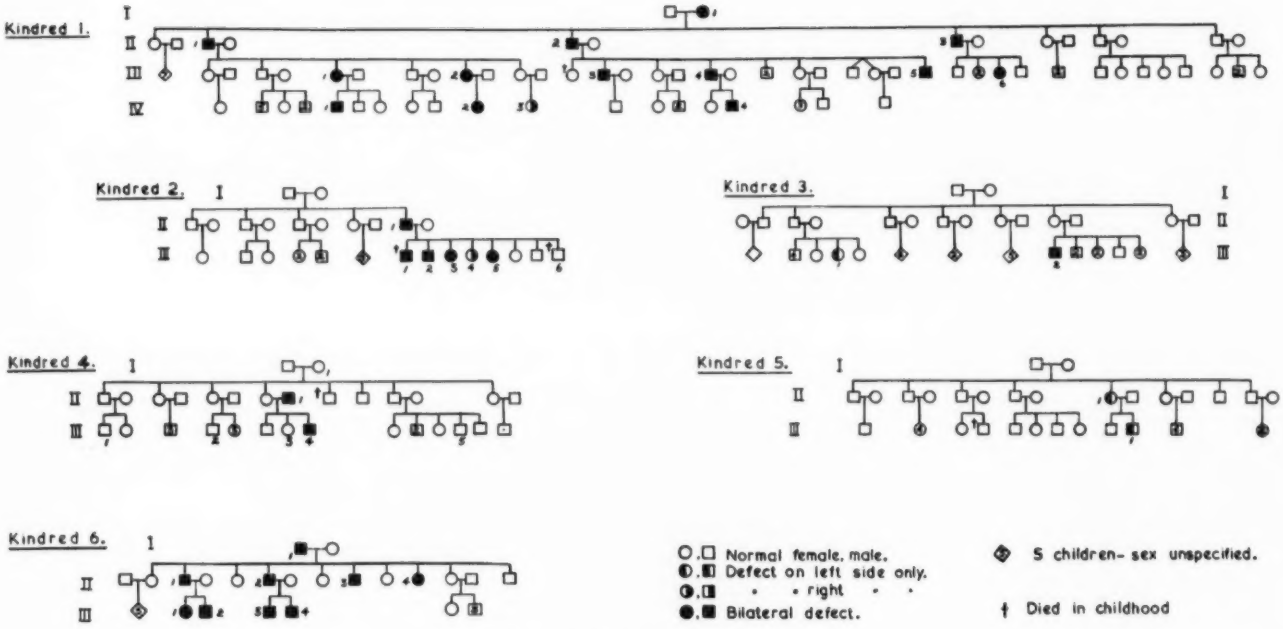

FIG. IV. Pedigrees of congenital ptosis.

Kindred 1

I. 1. Female. Bilateral ptosis, condition of parents unknown.
II. 1. Male. Bilateral ptosis.
II. 2. Male. Bilateral ptosis.
II. 3. Male. Bilateral ptosis.
III. 1. Female. Bilateral ptosis.
III. 2. Female. Bilateral ptosis with no trace of epicanthus.
III. 3. Male. Bilateral ptosis.
III. 4. Male. Bilateral ptosis, marked on left side, slight on right. No epicanthus.
III. 5. Propositus G-207. Male. Bilateral ptosis without epicanthus. More severe on right side. Right pupil half coloured.
III. 6. Female. Bilateral ptosis without epicanthus.
IV. 1. Male. Bilateral ptosis without epicanthus.
IV. 2. Female. Bilateral ptosis. No epicanthus.
IV. 3. Propositus G-165. Male. Moderate ptosis affecting right eye only. No epicanthus.
IV. 4. Propositus G-206. Male. Moderate bilateral ptosis, more severe on the right side. No epicanthal webbing. Condition relieved by operation.
III. 5. Propositus G-208. Female. Gross bilateral ptosis with marked epicanthus. Ptosis slightly more severe on left side. Relieved by operation.
III. 6. Male. Patent ductus arteriosus, died at 10 weeks of age. No ptosis or epicanthus.

Kindred 3

III. 1. Propositus G-210. Female. Ptosis of left eyelid. No epicanthus. Condition improved by operation.
III. 2. Bilateral ptosis with no epicanthus.

Kindred 4

I. 1. Female. Became blind at the age of 48 ("tumor").
II. 1. Male. Gross bilateral ptosis. Brought up in an orphanage, and has no knowledge of his parents.
III. 1. Male. Like III. 5., suffers from corneal opacity.
III. 2. Female. III. 2., and her three male sibs are all severely myopic.

FIG. V. Kindred 2.

III. 2. III. 4. II. 1. III. 5. III. 3.

Kindred 2 (Fig. V)

II. 1. Male. Moderately severe bilateral ptosis, slightly worse on left side. No epicanthal webbing.
III. 1. Female. Accidental death in infancy. Bilateral ptosis; whether epicanthus was also present is not known.
III. 2. Propositus G-214. Male. Moderately severe bilateral ptosis with slight epicanthus, relieved by operation.
III. 3. Propositus G-205. Female. Bilateral ptosis of moderate severity corrected surgically. Epicanthal webbing just detectable. Ptosis not quite as marked as in III. 2.
III. 4. Propositus G-204. Female. Gross ptosis of right eye, improved by operation. No epicanthus.
III. 3. Female. No ptosis, but has marked squint (right eye).
III. 4. Propositus G-259. Male. Bilateral ptosis without epicanthus, moderate on left side but severe on right.
III. 5. Male. See III. 1.

Kindred 5

II. 1. Female. Slight left ptosis. No epicanthus.
III. 1. Propositus G-260. Gross ptosis of left eyelid. No epicanthus.

Kindred 6

I. 1. Male. Severe bilateral ptosis with epicanthus. Both parents normal.
II. 1. Male. Fairly severe bilateral ptosis accompanied by epicanthal webbing.
II. 2. Severe bilateral ptosis without epicanthus.

II. 3. Male. Bilateral ptosis with marked epicanthus.

II. 4. Female. Bilateral ptosis with epicanthus.

III. 1. Propositus G-270. Female. Bilateral ptosis and epicanthus. Palpebral fissures rather short. Condition improved by operation.

III. 2. Propositus G-271. Male. Bilateral ptosis and epicanthus, alleviated surgically.

III. 3. Propositus G-266. Male. Gross bilateral ptosis with epicanthus, relieved by operation.

III. 4. Propositus G-267. Male. Severe bilateral ptosis with epicanthus. Condition improved by operation.

Consideration of the data in Table 1 shows that in general the penetrance of the dominant gene causing ptosis lies between 90 and 95 per cent. in both sexes. Particular pedigrees, for example, Kindred 3, may show an apparently lower penetrance, possibly due to the presence of modifying genes in these families.

If hereditary congenital ptosis is caused by a rare dominant gene, the great majority of affected individuals will be heterozygotes. Sibships with one parent affected should therefore contain equal numbers of affected (including carriers) and normal individuals. For simple ptosis ($\chi^2 = 1.29$, $p = 0.20$-0.30) this condition is fulfilled by the data of Table 1; for ptosis with epicanthus ($\chi^2 = 7.71$, $p = 0.01$) there is a significant excess of affected individuals. This may be due to the fact that many of the pedigrees were selected for a high incidence of the defect, but further pedigrees must be collected before a definite conclusion can be reached. The combined data ($\chi^2 = 0.26$, $p = 0.80$-0.90) are in close agreement with the expected figures.

TABLE 1

ANALYSIS OF SIBSHIPS WITH ONE PARENT AFFECTED OR KNOWN TO BE A CARRIER

Defect	*Affected*		*Unaffected*[1]		*Carriers*		*Overall Sex Ratio in Sibships*	*% Males Affected of all Males*[2]	*% Females Affected of all Females*[2]
	Males	*Females*	*Males*	*Females*	*Males*	*Females*			
Simple ptosis	70	50	91	59	7	4	1·49	45·8±3·9	47·8±4·7
Ptosis with epicanthus	40	27	23	17	1	1	1·42	62·5±6·1	62·2±7·2
Total	110	77	114	76	8	5	1·47	50·9±3·3	51·9±4·0

[1] Excluding identifiable carriers.
[2] Including identifiable carriers.

MODE OF INHERITANCE

The pedigrees described above are all consistent with the hypothesis that ptosis is caused by a dominant autosomal (i.e. not sex-linked) gene. Stuckley (1916) believed that a recessive was involved, but the pedigree which he gave clearly excludes the possibility of such a mode of inheritance in the particular family which he studied. Subsequent authors are agreed on the dominant nature of the gene concerned.

An analysis of the published pedigrees of ptosis with and without epicanthus, together with those reported here, is shown in Table 1. All apparently complete sibships for which one parent was effected or could be positively identified as a carrier have been included, detailed references will be given elsewhere.

Certain individuals may transmit ptosis to later generations without themselves showing the defect, though they must be carriers of the gene concerned (Kindreds 1 and 3). If the gene involved is a dominant, its effect should be manifested in such carriers; the estimated percentage manifestation of a gene in such circumstances is referred to as its *penetrance*.

Analysis of the published pedigree material using Haldane's (1936) method for detection of partial sex-linkage fails to reveal any evidence of the participation of such genes in the case of either simple ptosis or ptosis with epicanthus.

RELATION OF PTOSIS TO EPICANTHUS

Much attention has been given to a distinction between simple ptosis and ptosis with epicanthus, and the defects have usually been attributed to two separate but closely linked loci (see, for example, Gates, 1942).

It is commonly stated that ptosis with epicanthus affects twice as many males as females, whereas ptosis alone affects both sexes with equal frequency (Duke-Elder, 1952). There is no basis whatsoever for this differentiation. There are in fact more affected males than females in sibships showing either ptosis alone or ptosis with epicanthus (Table 1), but the proportion of males which are affected is not significantly different from the proportion of females affected. The appearance of more affected males is due to the fact that the sibships contain a preponderance of males, both normal and affected. This overall excess of males just fails to reach statistical significance at the 5 per cent. probability level in the case of ptosis with epicanthus ($\chi^2 = 3.3$, $p = 0.05$), but is significant in the sibships showing ptosis alone, and in the combined figures for the two conditions. A similar excess of males was observed by Steinberg *et alii* (1951) in their data on hereditary psoriasis.

Usher (1935) collected a number of pedigrees in which epicanthus of the same morphological type as that associated with congenital ptosis was inherited as an isolated condition. Epicanthus is, however, a normal condition in the foetus. While working in Munich, Martin (quoted in Gates, 1948) found that epicanthus was present in 33 per cent. of infants of one to six months of age; at three to six years 14 per cent. of males and 5 per cent. of females were affected, and at 12-25 years 3.3 per cent. of males and 2.6 per cent. of females. Epicanthus has not been found to occur by itself in pedigrees showing ptosis accompanied by epicanthus, whereas ptosis may occur without epicanthus in such families (as in Kindred 6).

It is not necessary, therefore, to postulate that a separate gene is responsible for the two different clinical manifestations; in each case the time of action during development, the mode of inheritance and penetrance are apparently the same.

The epicanthus accompanying ptosis in particular pedigrees is thus not necessarily due to a separate genetic system, but is rather a reflection of the retarded or unbalanced development caused by a gene which primarily affects the differentiation of the extraocular muscles. The consistent appearance of epicanthus with ptosis in some pedigrees and not in others may be caused by the modifying influences of elements of the genetic background common to members of a particular pedigree.

SUMMARY

1. It is considered that ptosis and ptosis with epicanthus are probably manifestations of the same dominant autosomal gene with high penetrance.
2. There is no significant difference in the proportion of affected males and females in sibships with one parent affected. The apparent difference in sex incidence of the condition is due to the high sex ratio observed in such sibships.
3. An appeal is made for the collection of unselected pedigree material for use in genetic studies.

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BICEPS CINEPLASTY FOR FOREARM AMPUTEES

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THE post-war development of upper limb prostheses has lagged sadly in this country. The notorious difficulties in supplying a workable terminal device have dampened the ardour of the limb fitter and the stimulus of an interested surgical profession has been conspicuously lacking.

Present interest was aroused by the progress reported from some American amputation services where cineplastic prostheses have been investigated and promoted on a fairly large number of patients. The purpose of this paper is to record with some enthusiasm a limited experience of this technique, whereby a terminal device is operated by a muscle motor constructed in the upper arm.

Historical Outline

The principle of utilizing some of the residual muscle mass of an amputation stump to activate a terminal prosthetic unit was first conceived and developed by continental surgeons. Various crude methods of attaching the prosthesis to the muscle remnants were suggested by Vanghetti (1898) and applied by Ceci (1905), but the current method of muscle canalization was developed by Sauerbruch (1916), Krukenberg (1917) and Putti (1919). The German clinics continued between the wars to develop the cineplastic technique with Lebsche's advance of dividing the insertion of the tunnelled muscle and the progressive use of more proximal (undamaged) muscles for motor units.

Kessler (1939) attempted, perhaps overenthusiastically, to popularize this method in America but it has only been accepted and developed on an official level in that country since the post-war report of Alldredge (1948) on the German wartime developments. Recent reports from Canty (1957) of the U.S. Naval Amputee Service and from Brav, Spittler, Luscombe, Kuitert, MacDonald, Vultee, Woodard, Fletcher, and Leonard (1957) of the Walter Reed Hospital indicate a general acceptance of biceps cineplasty in forearm amputees.

Cineplasty has not been popular in Britain, but in Australia important contributions were made by Rank and Henderson (1946) who developed an intricate hand prosthesis activated by forearm muscle motors. Owing to technical difficulties in its fitting, and a delicate unit with considerable upkeep costs, this prosthesis has not been continued since the war. Moreover the poor excursion of the forearm muscle tunnels and their weak power have been superceded by the less precise but much more powerful biceps motor.

These early applications of the cineplastic principle embraced the theoretical ideal of two muscle motors, one to open and one to close the terminal device. The introduction of spring closure into the prosthesis means that only one cineplasty is required with the advantages of more precise placement and more concentrated training. The other major advance in recent years has been the utilization of an uninjured muscle in the next proximal segment of the limb, thus eliminating the difficulties formerly met in scarred muscle bellies and amputation stumps of inconvenient length. Thus the biceps humeri is now used for forearm amputees and pectoralis major for short upper arm amputees or after shoulder disarticulation.

Selection of Patients

To avoid creating the impression that cineplasty is regarded as the answer to all amputation problems, its limitations must be clearly defined.

In forearm amputees, forearm muscle tunnels with their weak pull, short excursion, and intricate personalized prosthesis have at present been abandoned. Furthermore the presence of such distal tunnels interferes with the fitting of satisfactory sockets. Biceps humeri, when tunnelled provides a powerful motor capable of 50 to 80 pounds force with an excursion of over 2 inches, the forearm stump is left undisturbed and there is the

added advantage of replaceable terminal devices being available. For forearm amputees the biceps cineplasty has been a great advance, but it has little advantage over the conventional limbs in the long above-elbow or through-elbow amputees because of the difficulty in fitting a stable socket over a stump so close to the moving muscle motor. Pectoralis major is also utilized for short above-elbow amputees and for some with shoulder disarticulation, but the technical difficulties associated with fitting suitable prostheses are at present considerable. There would appear to be increasing scope for the pectoral cineplasty if used in conjunction with the normal shoulder harness prosthesis in these high amputees where it can be utilized to operate an elbow locking device.

From a limited personal experience of only 7 upper limb cineplasties comprising 6 biceps cineplasties and one pectoral cineplasty it is felt that a guarded optimism can be voiced if these limitations are recognized, and care is taken in selection of patients.

Blindness, the universal contra-indication to any prosthesis is recognized and the Krukenberg operation reserved for these unfortunates. Other contra-indications include those too young and those too old to learn the manipulation of a cineplastic device and those over-dependent personalities not uncommon in Southern European migrants. Heavy labouring cannot be performed because of wear and tear on the cables and moving parts. Also patients living at great distances from the limb fitters makes replacement of parts so tedious as to constitute a *contra-indication to cineplasty.*

Given a young, intelligent forearm amputee, a biceps cineplasty prosthesis can be recommended as superior to the orthodox harness type of prosthesis. At present, opinion is reserved on the use of either cineplasty for upper arm amputees.

BICEPS CINEPLASTY

In this procedure the effective insertion of the biceps humeri is transferred from the radius to a skin tunnel passing through the distal part of its muscle belly. To accommodate for this dramatic change in its function, and to increase the independence of cortical

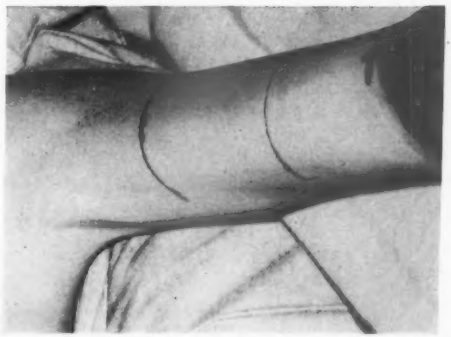

FIG. Ia. The skin flap is outlined, with lateral humeral epicondyle indicated by an arrow.

FIG. Ib. The skin flap has been tubed and lies in the foreground. The biceps has been divided from its insertion and the tunnel prepared through its muscle belly.

FIG. Ic. Lateral view of the completed operation showing the skin tube sutured to the outer wound margin and the muscle resurfaced with a thick dermatome graft.

control over this new function, intensive pre-operative and post-operative muscle training programmes are essential. These have been described in detail already (Gale and Hueston, 1957) and for at least two weeks before operation, exercises are used to increase muscle bulk and to begin this process of "cerebral isolation" of biceps for its future specialized function.

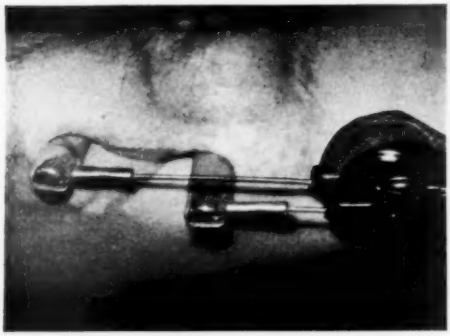

FIG. II. Double exposure photograph to show the excursion of the skin tunnel used in operating the prosthesis.

The operation

The method used is that followed by Spittler and Fletcher (1954) using a 3½ inch square skin flap based over the medial intermuscular septum 3 inches above the medial humeral epicondyle (Fig. Ia). This is tubed with the skin innermost. After isolation of the biceps, preserving its nerve supply, the tendon of insertion is divided and oversewn. A tunnel is made through the muscle belly immediately above the tendinous line, taking care to split rather than divide the muscle fibres, until 2 fingers are easily admitted (Fig. Ib). The skin tube is drawn through this muscle tunnel and sutured to the lateral wound margin with cranial rotation through a right angle to bring its suture line proximally and out of the future pressure area of the tunnel. A thick dermatome graft is used to resurface the exposed muscle belly (Fig. Ic).

Early resumption of the limb function is important and within two weeks of operation elbow movement is encouraged, without biceps participation. During the third week when the wound is healed, static contraction of the biceps is begun, often using simultaneous contraction of biceps on the sound side to help in early re-education. This extremely important phase of post-operative exercises, which includes muscle stretching, resisted contractions, independence exercises and also weight-lifting for the intact muscles has been described elsewhere along with a simple pulley used for the resisted exercises in this programme (Gale and Hueston, 1957).

FIG. III. A unilateral forearm amputee with a biceps cineplasty and voluntary opening split-hook.

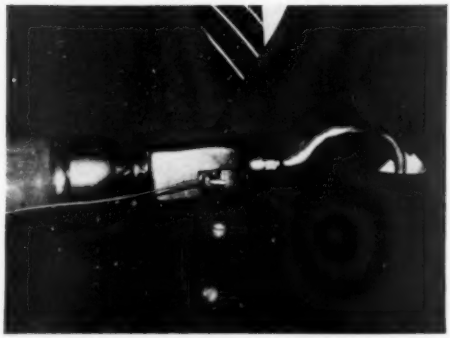

FIG. IV. The proprioceptive sense of the cineplasty combined with the fine tipped split-hook allows exploration of pockets.

The standard required before commencing to use a prosthesis is a pull of at least 15 lbs. through a tunnel excursion of 1½ inches (Fig. II). This is usually attained during the second month when the prosthesis may be fitted. For some months the normal use of the prosthesis does not suffice to keep up the strength and excursion of the muscle motor and regular resisted exercise periods each day are continued at home by the patient. Eventually a pull of 50 to 80 lbs. through 2 to 2½ inches is achieved.

The tunnel

In such a confined space as the muscle tunnel there is little escape for sweat and other skin secretions so that the lining skin becomes moist, greasy and even offensive unless scrupulously cared for. Washed through twice daily it is dried with gauze and spirit. Powder tends to cake and so increase irritation and is best avoided. Plain soap and water is the safest cleansing agent although "pHisoHex" is sometimes substituted and the patient continues this regular toilet at least twice daily. Ulceration in the tunnel may follow too vigorous exercise, the use of steel pins instead of acrylic, too thin a pin, or neglect of cleansing of the tunnel skin. Zinc cream and resting the tunnel allows healing and the cause of breakdown is thereafter avoided.

The prosthesis

A straight acrylic rod fitting the skin tunnel is connected by 2 Bowden cables to a common terminal lever on a split-hook. This is attached to a socket of laminated fibre glass and polyester resins which is stabilized by a simple epicondylar cuff across the cubital fossa.

Two types of split-hook have been used — a voluntary opening and a voluntary closing type. The voluntary opening type with spring closure of two adjustable strengths, namely 3½ and 7 pounds, is simple to operate (Fig. III), but limited in the sensitivity of grip as the muscle needs to hold the hook open to prevent the crushing of delicate objects such as a cigarette. This proprioceptive sense is important in the terminal device and is better achieved with cineplasties than with orthodox prostheses. By continuous muscle activity the spring tension of the voluntary opening hook can be varied to supply some idea of "feel." This however, is inferior to the quality of the proprioceptive sense achieved by the voluntary closing device in which a cam clutch type of locking mechanism is incorporated so that no muscle action is needed to hold objects fragile or heavy, once they are in the hook. For this reason, and also because it is near to normal physiologically, a voluntary closing split-hook has been used in all cases except one. This is the Army Prosthetics Research Laboratories hook.

FIG. V. The cosmetic advantage of a hand prosthesis is outweighed by the difficulty of picking up from a flat surface and its obstruction of the patient's view; here compared with the split-hook.

A hand prosthesis is available in which the thumb and the next two fingers are the movable elements with a "chuck" grip, but no patient has preferred this to the split-hook except for important social occasions. This preference for the utility split-hook is based on its versatility which in turn is due to the fine grip allowed by the hook tips. Pockets can be explored with a split-hook (Fig. IV), but not with a hand prosthesis; small objects can be picked up with confidence, papers turned over and eating utensils handled with greater ease by the split-hook.

Finally the split-hook does not obscure the view of small objects when being picked up as does the more massive "hand" prosthesis (Fig. V). Despite its initially offensive appearance, the split-hook terminal device is

universally the more favoured, unless the patient's occupation involves meeting the public on a large scale when he may decide to sacrifice some function for improved appearance. In practice every patient is provided with both a "hand" and a "split-hook" which are interchangeable.

One patient with bilateral forearm amputations has literally no social disability with bilateral biceps cineplasty and voluntary closing split-hook prosthesis (Fig. VI). This man can conduct his normal life, requires no special provision for eating or drinking, and has recently obtained his driving licence in Melbourne, all of which indicate the degree of proficiency he has obtained.

FIG. VI A bilateral forearm amputee with bilateral biceps cineplasty. No special provision is necessary for eating or drinking.

ADVANTAGES OF BICEPS CINEPLASTY

The most obvious advantage of these prostheses is the freedom from the encumbrance of shoulder harness. The increased comfort particularly in hot weather, but also in daily assembling of the mechanism which follows the elimination of shoulder harness, is most striking (Fig. VII).

There is an increased range of activity with biceps cineplasty, particularly above the level of the shoulders whereas the orthodox harness-type prosthesis becomes inefficient when the arm is elevated because the harness rides up and can no longer exert effective traction. The biceps muscle motor is efficient in all positions of the arm and enables the placing of objects on high shelves and other higher reaches of activity.

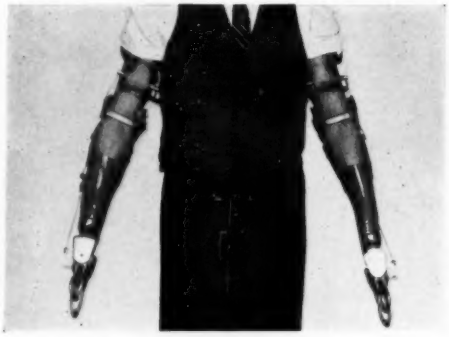

FIG. VII. The same patient as Fig. VI showing the freedom from shoulder harness.

The increased proprioceptive sense achieved by the voluntary closing mechanism through the biceps muscle tunnel has already been discussed. Greatly improved precision of control over the terminal prosthesis has followed the use of the voluntary closing split-hook where the whole range of strength of the active muscle contraction can be directly applied.

The complete independence of each arm in bilateral amputees is an improvement over the relatively more cumbersome bilateral shoulder harness mechanism required with the conventional prosthesis.

SUMMARY

The present trends in the application of the cineplasty principle are outlined indicating the current use of a muscle motor in the next proximal segment of the arm.

In forearm amputees the biceps cineplasty is found to give more precise control and an increased sphere of activity to the terminal prosthesis.

The elimination of the shoulder harness allows greater comfort and, in a bilateral amputee, complete independence of each limb.

The technique and management of biceps cineplasty is outlined. However the important factors in securing a useful cineplasty lie not in the surgery but in

(a) selection of an intelligent, co-operative patient,

(b) a thorough programme of pre- and post-operative physiotherapy,

(c) a light and comfortable prosthesis, and

(d) full rehabilitation until a normal life and occupation are resumed.

ACKNOWLEDGEMENTS

The first three of the biceps cineplasties were performed by the author at the Repatriation General Hospital, Heidelberg, and it is a pleasure to acknowledge the help provided at all stages of their treatment in that institution.

The team work necessary in the care of these patients was enthusiastically provided by Miss A. Gale, physiotherapist at the hospital, Dr. R. Klein at the Repatriation Artificial Limb Factory, and of course by the spirit of the patients themselves.

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SMALL BOWEL OBSTRUCTION FOLLOWING ABDOMINO-PERINEAL EXCISION OF THE RECTUM

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IN a personal series of 287 patients who had abdomino-perineal excision of the rectum for malignant tumour, 11 (3.8 per cent.) developed a small bowel obstruction soon after operation which was responsible for the death of the patient or an emergency laparotomy. In 6 of these eleven patients the obstruction was caused by portion of the small bowel herniating through the pelvic peritoneal suture line (Fig. I). These 6 cases have been analysed in this communication. In a seventh case, not included in this series, the small bowel was adherent to the site of the ligature of the inferior mesenteric vessels and it was thought that a knuckle had herniated through the upper end of the suture line.

Goligher, Lloyd-Davies and Robertson (1951) reviewed patients complicated by small bowel obstruction following excision of the rectum at St. Mark's Hospital. There were 37 cases in a series of 1,302 (an approximate incidence of 3 per cent.). In 12 the obstruction arose in connection with the pelvic peritoneal suture line, but in only one was there a gap through which a knuckle of small intestine had herniated. Gabriel (1948) observed that the complication had occurred twice in 650 perineal excisions, but he had not observed it after the perineo-abdominal method. McCune and Keshishian (1953) had one such case in their series of post-operative intestinal obstructions. Butler (1952) reported the loss of one patient in a series of 154 combined excisions in whom bowel obstruction was produced by adhesion to the pelvic floor.

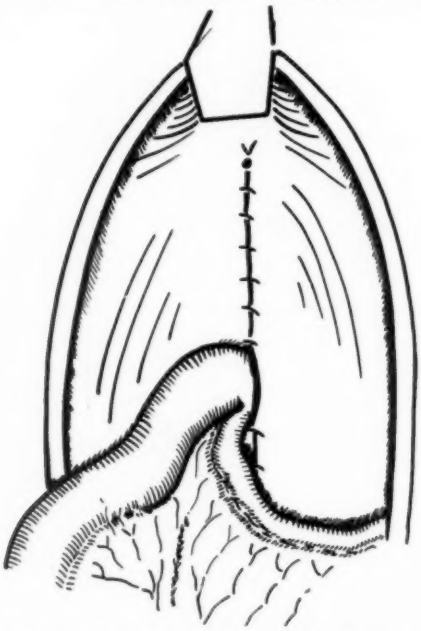

FIG. I. (i) Small bowel partially herniates through a defect in the peritoneal suture line.

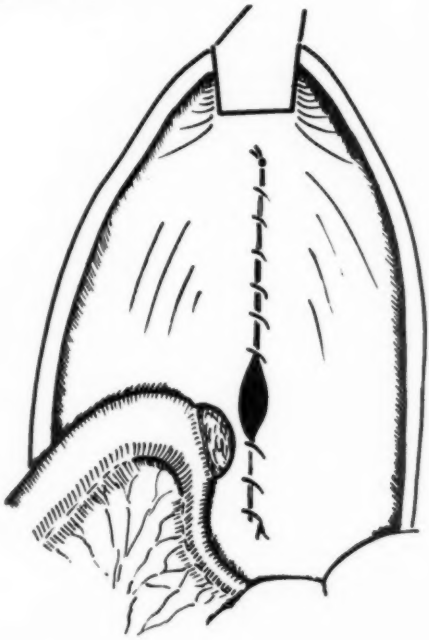

FIG. I. (ii) After reduction the strangulated portion of bowel has the appearance of a diverticulum.

Case histories of patients with small bowel obstruction caused by herniation of the small bowel through the suture line.

Case 1

Mr. T.Y. (R.M.H. No. 150,533), aged 82 years, and suffering from a carcinoma of the anus, had a synchronous combined excision of the rectum, anal canal and anus on the 19th November, 1953. His condition was satisfactory for twenty-four hours. On the second post-operative day his temperature rose to 103° Fahrenheit, and after this he deteriorated slowly. He did not complain of pain, did not vomit, and at first his abdomen showed minimal distension. He gradually became confused, disorientated and finally he died on the 30th November, eleven days after operation. Autopsy showed that a knuckle of terminal ileum had herniated through the pelvic peritoneal suture line, with considerable distension of the small bowel proximal to it.

Case 2

Miss I.D. (R.M.H. No. 150,926, aged 26 years, suffering from a carcinoma of the rectum, had a synchronous combined excision of the rectum on the 8th December, 1953. She tolerated this operation well and her condition on the first post-operative day was excellent. On the second day she complained of abdominal pain, but as there was no vomiting and no distension it was thought that she was unduly sensitive to pain. Further, her temperature rose to 102° Fahrenheit, which seemed to indicate an infection. However, the pain persisted and gradually she became distended. Operation was performed on the 15th December. A knuckle of terminal ileum was found herniated through a hole in the pelvic peritoneal suture line; there was considerable distension of the small intestine above the site of the obstruction. The bowel was reduced and the defect closed, but intervention was too late and she died on the 18th December, ten days after the original operation.

Case 3

Mr. N.K. (R.M.H. 157,193), aged 61 years, suffering from a carcinoma of the rectum, had an abdomino-perineal excision of the rectum on the 15th October, 1954. He tolerated the operation well and his condition on the first post-operative day was good. On the second post-operative day he vomited profusely and a gastric tube was passed. His temperature rose to 102° Fahrenheit and on the following day he became seriously ill. He remained in this state for two days before his abdomen became distended. By this time he was too sick for further intervention and he died on the 25th November, ten days after the original operation. Autopsy revealed the ileum, 30 centimetres from the ileo-caecal junction, firmly adherent to the pelvic suture line and further dissection showed that this was caused by herniation of a knuckle through a defect in the pelvic suture line. The small bowel was distended considerably above this level.

Case 4

Mr. A.P. (R.M.H. 160,103), aged 48 years, suffering from a carcinoma of the rectum, was subjected to abdomino-perineal excision of the rectum on the 15th March, 1955. His condition on the first post-operative day was good, but on the second he complained of continuous severe pain in the abdomen. His temperature rose sharply to 103° Fahrenheit. On the third day he was distended. On the fourth day (19th March) he vomited and operation was performed. A knuckle of the terminal ileum had herniated through the pelvic peritoneal suture line. The bowel was reduced and the defect closed. He recovered slowly and has remained well since (four years).

Case 5

Mr. G.T. (R.M.H. 170,503), aged 28 years, suffering from a carcinoma of the rectum, was subjected to abdomino-perineal excision of the rectum on the 27th August, 1956. His condition was very satisfactory for two days after which he complained of pain and gradually became distended. He had no vomiting until the eighth post-operative day. Laparotomy was performed in the 8th September, and a knuckle of ileum was found herniated through the pelvic suture line. Proximal to this level the bowel was distended and distal to it, collapsed. The bowel was reduced, and the defect repaired. His convalescence was uneventful and he has since remained well (two and a half years).

Case 6

Mr. McK. (private case referred by Dr. W. Austin Cooper), aged 59 years, with a carcinoma of the rectum, was submitted to abdomino-perineal excision on the 6th February, 1957. From the first post-operative day he complained of severe abdominal pain. His temperature rose to 103° on the second post-operative day, and he became drowsy. He improved a little on the third and fourth post-operative days, but on the fifth he became distended and operation was performed on the 11th February. A knuckle of terminal ileum was found herniated through the pelvic peritoneal suture line. The bowel was distended to a considerable size. The hernia was reduced, and the defect closed. His convalescence was interrupted by the development of a lung abscess, which required drainage. Subsequently he regained health and he has remained well (two years).

DISCUSSION

In this series herniation of a knuckle of small bowel through the pelvic peritoneal suture line was responsible for just over half the cases of small bowel obstruction following abdomino-perineal excision of the rectum. Three of the 6 patients with this complication died, and the 3 survivors were gravely ill before recovery was complete.

Aetiology of complication

The complication is due to a hole in the pelvic peritoneal suture line. In each of the 6 cases reported here the defect appeared immediately after operation because symptoms were present on the first or the second

post-operative day. In each of these cases a continuous suture was used to close the peritoneum; this was commenced anteriorly and continued posteriorly to the pedicle. If the peritoneum is thin, or is sutured under tension, it is likely to tear and cause a hole; whilst if the suture material used for the continuous suture is not held firmly by an assistant, a defect is almost inevitable.

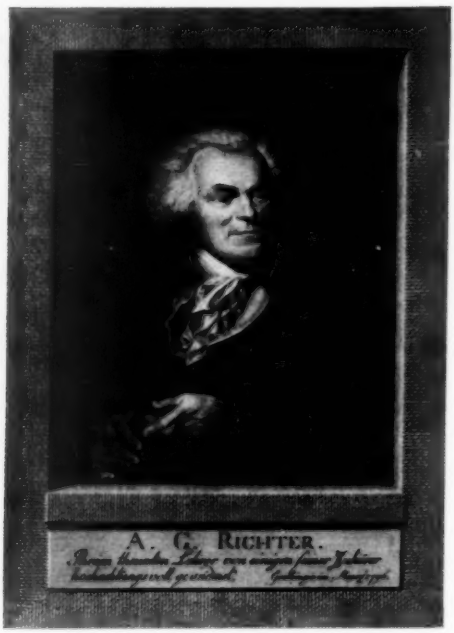

FIG. II. August Gottlieb Richter (1742-1816). (By permission, Wellcome Historical Library.)

The herniated portion of the bowel is drawn into a pseudo-diverticulum. Alexis Littré, in 1700, described a hernia which was later regarded as containing a Meckel's diverticulum. Littré thought that the diverticulum had been acquired (Keynes, 1956). In 1785, Richter (Fig. II) gave an account of herniae in which only part of the bowel was strangulated, and he appreciated the fact that such ruptures may be found anywhere in the abdominal cavity. The herniae described in this series are identical with those of Richter and perhaps also with those of Littré.

Clinical features of the complication

A Richter's hernia is always liable to cause symptoms which are difficult to interpret, but occurring immediately after an operation of this magnitude the difficulties are enhanced. In this series, unusually severe, constant abdominal pain was observed on the second post-operative day in four of the six cases; in the fifth, an elderly patient (Case 1) pain was not a feature at any time, and in the sixth (Case 5) symptoms appeared on the third post-operative day. Distension did not develop until several days later. Vomiting was an early feature in only one case (Case 3). A curious feature in five of the six cases (with the exception of Case 5) was the sharp elevation of the temperature on the second post-operative day, in one case to 103.2° Fahrenheit.

Diagnosis of complication

Severe abdominal pain on the second post-operative day, especially if associated with a sharp rise of temperature, should lead to suspicion that such a complication has supervened. In Case 1, the diagnosis was not considered. It was felt that his age was in some way responsible for the unsatisfactory progress and deterioration. In Case 2, laparotomy was not performed until it was too late. The patient was very young and apprehensive and at first it seemed that she complained unnecessarily; later when she became distended it was thought that her colostomy was too tight. In Case 3, the patient deteriorated so quickly that laparotomy was considered too dangerous and conservative treatment was continued in the hope that it would lead to some improvement.

Treatment of complication

Laparotomy will reveal the distended small intestine. Further examination will show it adherent to the pelvic floor. When the small bowel is separated a small hole will be found. The knuckle of bowel is congested, but in none of the cases in this series was it necessary to resect the affected portion of the small bowel. The defect is closed with interrupted catgut sutures, but care should be taken not to take too wide a bite because the ureter is often drawn towards the midline by the suture apposing the edges of the peritoneum.

Prevention of the complication

A single continuous suture seems likely to lead to this complication (Fig. III). The peritoneum must first be stripped off the bladder and the side walls of the pelvis

(Lawrence Abel's "strip-tease" manoeuvre) so that the suture line is free from tension. Goligher, Lloyd-Davies and Robertson (1951) favoured suturing the peritoneum in such a way as to bring the serous edges into apposition, turning the free edges downwards towards the pelvic cavity. Abel (1957) advises a similar procedure using a continuous interlocking stitch. The presence of a raw peritoneal edge has not proved important in this series and it is now the writer's practice to employ a simple continuous interlocking suture reinforced at intervals by an interruptured suture (Fig. IV).

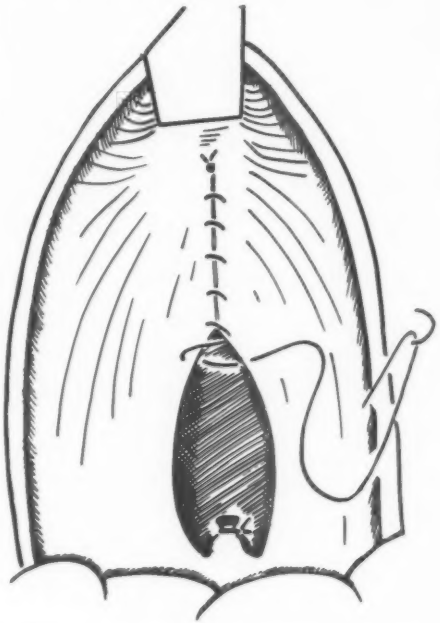

FIG. III. Usual method of suturing pelvic peritoneal flaps after combined excision of the rectum.

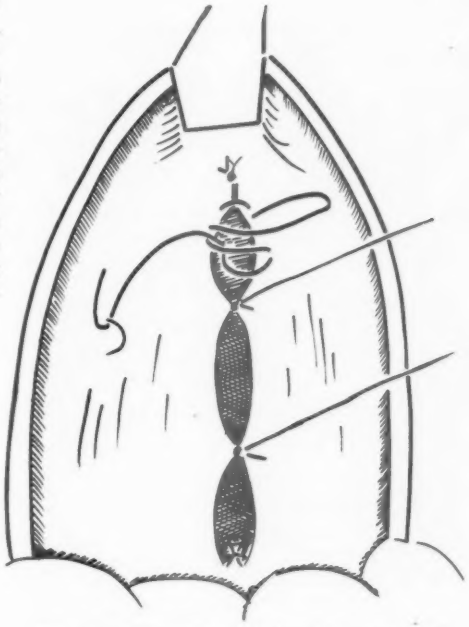

FIG. IV. Recommended technique for suturing the pelvic peritoneal flaps. An interlocking continuous catgut, and several interrupted sutures are inserted.

SUMMARY

1. A series of 6 cases subjected to abdomino-perineal excision of the rectum and complicated in the immediate post-operative period by herniation of a knuckle of small intestine through the pelvic peritoneal suture line is recorded.
2. The complication is due to a defect appearing immediately after operation because symptoms appeared on the first or second post-operative day.
3. The most important symptom was pain on the second post-operative day. This was often associated with a rise in temperature which was misleading.
4. The complication is likely to be overlooked because symptoms are not typical of obstruction until relatively late.
5. The complication can be prevented by attention to suturing of the pelvic peritoneum. A continuous interlocking suture reinforced with interrupted sutures is recommended.

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MICRODISSECTION STUDY OF LATE RENAL DISEASE

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"There are challenges of a salutary severity that stimulate the human subject of the ordeal to a creative response; but there are also challenges of an overwhelming severity to which the human victim succumbs". Arnold J. Toynbee, "A Study of History", Vol. 2, Part II, D, vii.

A SERIES of cases of non-specific renal disease, being investigated by microdissection technique, ranged from the macroscopically normal to those showing obvious and extreme change. One of the former has been described (Baxter, 1959) and, in contrast, an example of the second type is presented here.

Organs which are macroscopically normal with negligible histological change may be demonstrated to have undergone modifications due to chemical or bacterial injuries which were not clinically manifest; these therefore can be regarded as "wear and tear", regeneration being a prominent feature. Those exhibiting gross macroscopic and extreme histological alterations, on microdissection would be expected to, and do, show profound changes in which degeneration predominates.

When severe injuries have been repeated, the degree of parenchymal damage may be such that no recognizable structure, other than connective tissue, remains. This would commonly be so but for the perennially astonishing capacity of almost all tissues of the body for repair and regeneration. This phenomenon, the fundamental significance of which is implicit in most treatments and all favourable prognoses, may be observed in all but the severest injuries. Even in the present case, where parenchymal damage has resulted from an overwhelming infection (or series of infections), this is still demonstrable.

HISTORY AND MATERIAL

The specimen was obtained at operation from a male aged 25. The patient was admitted to hospital with a six-months history of pain in the right loin and, occasionally, under the right costal margin. The pain was so severe that the patient was prevented from working.

On examination, there was slight tenderness in the right loin but no other abnormality was detected. Blood pressure was 120/85 mm. of mercury.

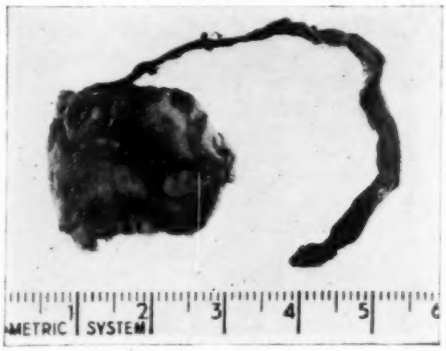

FIG. I. Photograph of the atrophied right kidney and attached ureter.

INVESTIGATIONS

Urine showed no microscopic abnormality and blood urea was 40 mg./100 ml. Right retrograde pyelogram was performed on three occasions. The catheter could be passed only half-way along the length of the ureter. Aortogram revealed a large left kidney, no right kidney being seen. Perirenal air insufflation outlined the left kidney but no right kidney was demonstrated. At operation a small right kidney was found and right nephrectomy was performed. When received, the specimen was fixed in formolsaline and was a dark grey colour. The organ was flattened and measured 2.5 x 2.0 x 0.5 cm. The segment of attached ureter was approximately 5 cm. in length and the proximal portion was considerably thinner than the distal part (Fig. I). The renal capsule was thickened and "shaggy" in appearance, as was the surface of the ureter.

METHODS

One half of the kidney was removed for histological examination; the remainder was used for microdissection. Appropriate portions were incubated at 19°C. for eighty-four hours in concentrated hydrochloric acid, following the technique of Oliver, MacDowell and Tracy (1951). When cutting the segments for incubation, the material was found to be tough and firm in contrast with the normally soft renal tissue. The long maceration time — usually about half this period is sufficient— was necessitated by the degree of fibrosis.

After incubation, the material was dissected by hand under a stereoscopic microscope, working at magnifications of 50 and 100. The material photographed was removed from the dissecting dish and mounted in a drop of water on a glass slide. All photographs of microdissected material are of unstained tissue, made with Leica camera and adapter using 35 mm. Ilford Micro-Neg. Pan film.

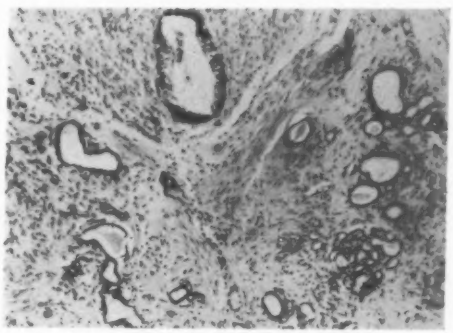

FIG. II. Photomicrograph of a section (low power) showing connective tissue in which are present tubules or ducts and groups of diverticula (cf. Figs. XVIII and XIX). These are lined by small cuboidal cells, containing deeply-staining nuclei. (x 95)

RESULTS

Histological examination

In the sections stained with haematoxylin and eosin, the thickened fibrous capsule surrounding the organ appeared to be well vascularized and, at times, exhibited frank haemorrhage. There were also areas of haemorrhage in the perirenal adipose tissue presumably due to the trauma of removal.

Under low power the tissue was a large area of fibrous connective tissue in which parenchymal units of various sizes were scattered in a random fashion. Parts of the connective tissue were extremely fibrous and relatively acellular.

It was not possible to distinguish cortex and medulla, the whole having a relatively uniform appearance. Glomeruli were not present; the tubular remnants ranged from small groups of 6 to 8 cells to giant cystic structures measuring up to 630μ in diameter.

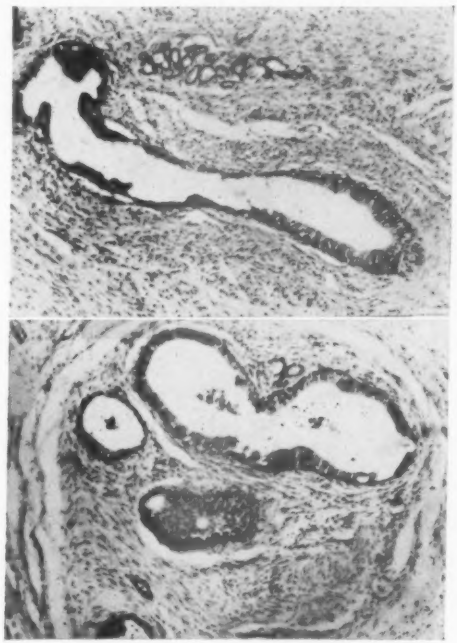

FIG. III. Photomicrograph of section showing dilated tubules within a concentric connective tissue and muscle meshwork and collections of small "tubules" (cf. Fig. XVIII). One tubule shows hyperplasia of lining cells with almost obliteration of the lumen. (x 95)

Many of the tubules, both small and large, contained a pink homogeneous substance. In addition, some tubules contained desquamated and inflammatory cells and there were foci of calcification within the tubular walls and also in the lumina. There were collections of small tubules or ducts lined by small cuboidal cells containing deeply staining nuclei (Fig. II).

Some tubules were lined by a single row of low cuboidal epithelium whilst others con-

tained several layers. Some of the larger tubules were lined by a columnar epithelium and, at times, this also was multilayered. In other instances the cells were heaped up at random within the lumina, occasionally almost causing obliteration (Fig. III). The ducts of Bellini were lined by tall columnar cells which, once again, were several layers in thickness, or heaped up in localized areas.

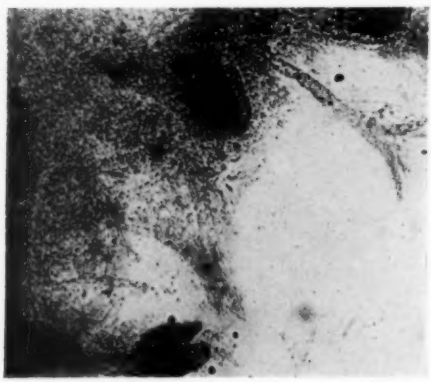

FIG. IV. Microdissection photomicrograph showing "ghost glomeruli" embedded in a connective tissue meshwork. Branching blood vessel also present. (x 56)

FIG. V. Photograph showing remnants of branching blood vessel with atrophied glomeruli. (x 95)

Throughout the tissue there was a patchy infiltration of small round cells, plasma cells and polymorphonuclear leucocytes. Many of the smaller blood vessels showed a perivascular cuffing, whilst there were other widely-patent thin-walled vessels packed with erythrocytes.

With PAS stain the homogeneous material within many of the tubule lumina was positive. With Alcian blue a small proportion of the tubules showed the presence of mucin. Picro-Gomori and Masson's stain demonstrated that the connective tissue was largely collagenous and that there were muscle fibres present, mainly arranged in a concentric fashion round tubules or groups of tubular elements.

Microdissection

Even after eighty-four hours acid maceration an abundant amount of connective tissue persisted. During maceration a black adherent substance, which was firmly attached to the tissue but the nature of which was not determined, was dissolved. Until this was cleared it was impossible to proceed with dissection.

No functional glomerulus was observed but, rarely, enmeshed in the connective tissue, dark outlines resembling the pattern of glomeruli attached to the branches of the vascular tree, were demonstrated. Some very small "ghost glomeruli" were noted, but, among the larger ones, diameters ranging from 140μ to 270μ were measured (Figs. IV and V).

FIG. VI. A collecting tubule showing gross distortion from dilatation and constriction. Maximum diameter 500μ; minimum diameter 50μ. (x 24)

No specific part of the tubular system, with the exception of the collecting tubules, was recognizable as such with certainty. The majority of the collecting tubules observed showed gross distortions, massive dilatations alternating with constrictions and diameters ranging from the extremes of 50μ to 500μ,

were recorded in one segment (Fig. VI). Frequently this phenomenon resulted in a block-like segmental appearance in the tubule (Figs. VII and VIII).

FIG. VII. Portion of grossly hypertrophied collecting tubule showing segmental character. (x 95)

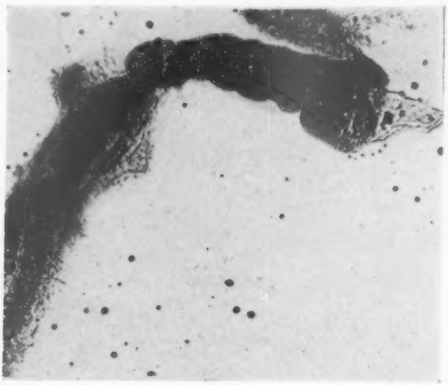

FIG. VIII. Collecting tubule illustrating the segmental nature. Maximum diameter 180μ; minimum diameter 35μ. (x 80)

Sometimes, associated with these tubules were branches assuming almost an "obelia-like" form as well as diverticula which ranged from minute to massive proportions; these occurred either solitarily (Fig. IX) or in groups (Fig. X).

FIG. IX. Collecting tubule, focussed to show its outline with a small single diverticulum. Maximum diameter 290μ; minimum diameter 15μ at the finest branch. (x 75)

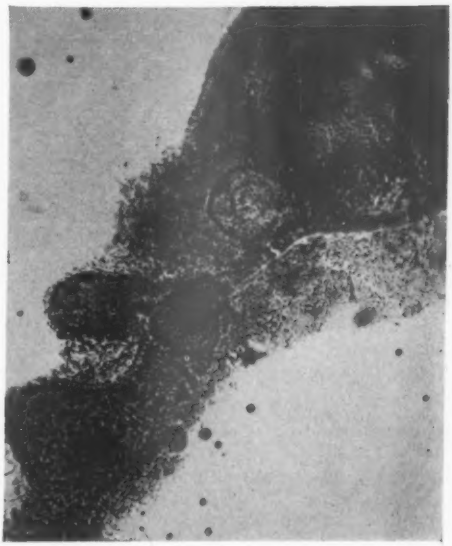

FIG. X. Portion of collecting tubule to show small diverticula. Note variable diameters of the tubule. (x 95)

Occasionally the tubular remnants were sealed at both ends (Fig. XI) and often branches were also involved (Fig. XII). These sealed remnants occasionally resulted in cyst formations and they were either attached or unattached to a tubular stalk (Figs. XIII and XIV).

At times, material was observed within the lumina of the tubules (Fig. XV). Occasionally intra-tubular material occurred in the area of dilatation (Fig. XVI and XVII).

FIG. XI. Tubule length, sealed at both ends. Maximum diameter 575μ; minimum diameter 195μ. (x 24)

FIG. XII. Sealed branching remnants of a collecting tubule exhibiting gross hypertrophy. Dimensions of branches—
(1) 565μ x 235μ.
(2) 320μ x 310μ.
(3) 180μ x 140μ. (x 95)

The small groups of tubules seen in the histological section were shown by dissection not to be separate tubules but rather small diverticula which, in the majority of instances, were flattened structures, almost paper-thin and were entirely dependent on the enveloping connective tissue meshwork for support. When teased from this tissue, they collapsed and disintegrated, the debris floating away (Figs. XVIII and XIX). Diameters up to 330μ were recorded for these flattened structures which occurred most frequently in grape-like clusters.

FIG. XIII. Small solitary tubular cyst. Dimensions 740μ x 1040μ. (x 48)

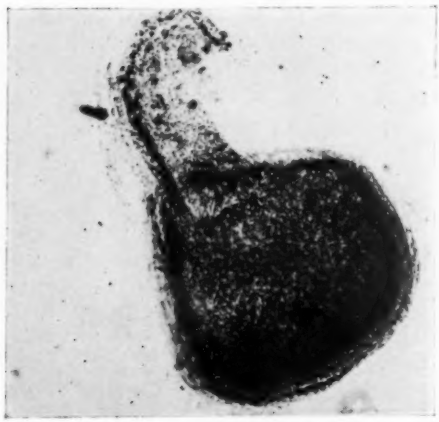

FIG. XIV. Small cyst with tubular stalk. Dimensions 395μ x 395μ. (x 100)

Often in a heavily diverticulated tubule, constriction was observed alternating with dilatation; diameters of one tubule ranged from 20μ to 150μ (Fig. XX). At other times, constriction had resulted in actual division of the tubule, while separation of diverticula was also proceeding, resulting in many small tubular remnants (Fig. XXI). A further step in the process was the complete separation of

diverticula to form segregated units while the adjacent tubule underwent still further subdivision (Fig. XXII).

DISCUSSION

The problem of the nature of the unilateral grossly contracted kidney is a difficult one which has been the subject of many studies. From these, diametrically opposed conclusions have been reached. It seemed appropriate to attempt to apply the microdissection technique to this question in an endeavour to illuminate it further. A brief review of the more recent literature emphasizes both the limitations of present methods of study and the degree of divergencies of opinion on the pathogenesis of these contracted organs.

FIG. XV. Branches of a collecting tubule showing dark material within the lumina. (x 95)

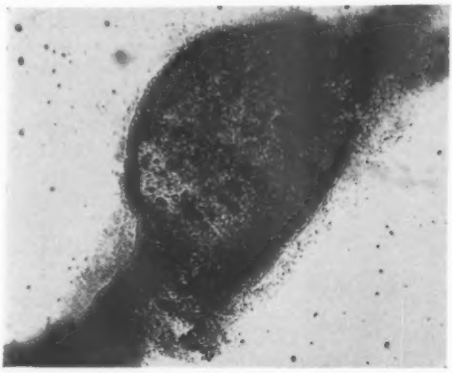

FIG. XVII. Gross tubular dilatation. Contents within the area of dilatation include cells as well as detritus. Maximum diameter 415μ; minimum diameter 35μ. (x 100)

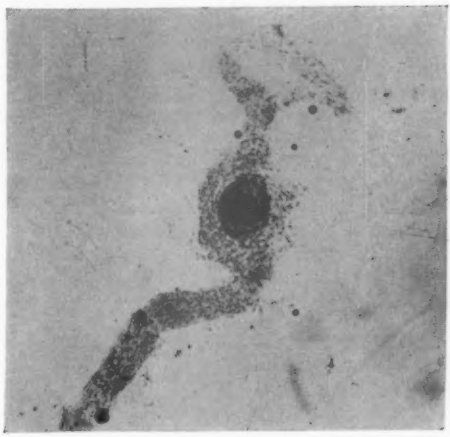

FIG. XVI. Portion of collecting tubule showing gross local dilatation with dark material within the dilated area. Maximum diameter at dilatation 130μ; minimum diameter behind dilatation 55μ. (x 95)

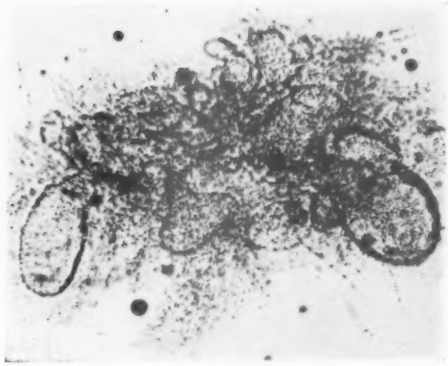

FIG. XVIII. Collection of small diverticula in supporting connective tissue; these are the sole remnants of the tubules in this area. Maximum diameters 205μ x 160μ; minimum diameters 35μ x 30μ. (x 75)

Until the early part of this century, that this kind of condition was the result of antenatal disease was generally accepted; furthermore, this was designated "congenital" and this, by implication, was hereditary or genetic in nature. The importance of acquired or environmental factors has been appreciated only relatively recently. It is recognized that pyelonephritis with atrophy may give an end picture of a "small diminutive renal mass" (Ritter and Kramer, 1950).

Emmett, Alvarez-Ierena and McDonald (1952) used the term "chronic atrophic pyelonephritis" in a discussion of 79 surgical cases. From this group, owing to the frequency of renal atrophy following "obstruction with infection and subsequent hydronephrotic atrophy", they excluded all cases in which obstruction or hydronephrosis could be demonstrated. They concluded that the unilateral "hypoplastic or atrophic kidney represents several different and distinct pathological entities"—the lesion may be congenital, acquired or a combination of the two conditions.

A plea is entered here for the cessation of the ambivalent use of the term "congenital". Conditions may be hereditary (genetic) or acquired (environmental)—an aetiological concept—on the one hand and antenatal (congenital) or postnatal—a temporal concept—on the other. The unnecessary confusion of the last few decades due to an uncritical intermingling of the two concepts in this term "congenital" will be actively discouraged by all thoughtful workers.

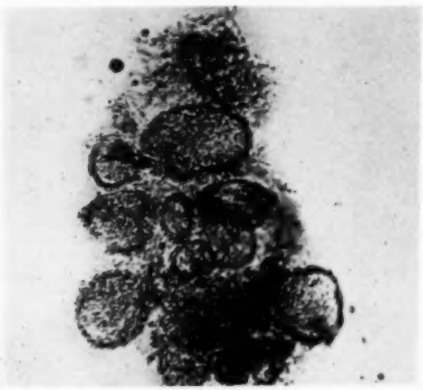

FIG. XIX. Small group of diverticula in connective tissue. Larger diverticula — dimensions 170μ x 105μ and 140μ x 122μ. (x 95)

FIG. XXI. Short length of collecting tubule, showing segmentation of tubule and the beginning of isolation of the individual diverticula. (x 95)

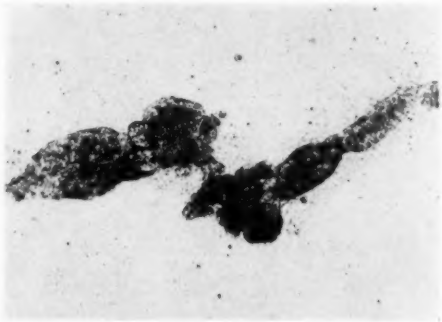

FIG. XX. Tubule showing irregular dilatation, constriction and multiple small diverticula. Maximum diameter 150μ; minimum diameter 20μ. (x 66)

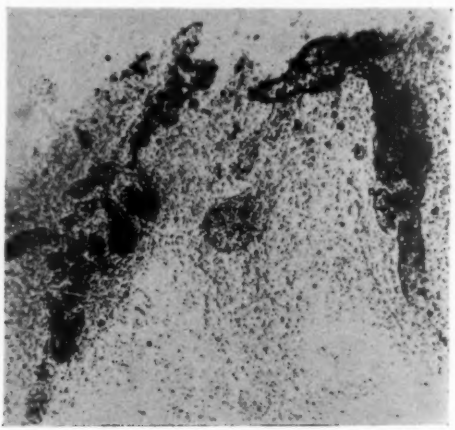

FIG. XXII. Pattern of diverticulated tubules showing through the connective tissue meshwork. Note in one tubule the disappearance of the main trunk and the persistence of the diverticula as independent units. (x 95)

Many of the cases dealt with by Emmett *et alii* (1952) were regarded as due to chronic atrophic pyelonephritis but they found no certain means of distinguishing this from those antenatally hypoplastic kidneys with a superimposed chronic inflammation. Most writers (Ritter and Kramer, 1950; Emmett

et alii, 1952; Marshall, 1953, and Burkland, 1954) agree that, in the terminal stage, differentiation is difficult "clinically, roentgenologically or histologically" (Ericsson and Ivemark, 1958).

Where gross abnormalities, clearly of antenatal (congenital) type, are present in parts of the urino-genital system, it is reasonable to conclude that there may be defects in the renal parenchyma (Marshall, 1953). In their absence aetiological classifications of late renal changes must be made with extreme caution. Marshall (1956) concluded that renal scars, excluding old infarcts, irrespective of association with hydronephrosis or atherosclerosis and independently of size or number (that is, whether single or multiple) have a common origin "in foci of maldeveloped tissue, and that their high incidence indicates that the kidney is frequently affected by a disorder of the congenital-developmental group". He stressed the vulnerability of dysplastic tissue to disease processes which had previously been given the immediate causative role. He discounted attacks of nephritis and pyelonephritis as aetiological factors in one series since he thought that a scar incidence of 62 per cent. was impossibly high; such a proportion of patients could not have had attacks of sufficient severity to result in permanent scarring. Apparently he dismissed as unworthy of consideration the possibility of repeated attacks of inflammation of subclinical intensity. This epitomizes one viewpoint.

Such an opinion ignores the large amount of information on the destructive effects of chemical and bacterial agents and experimental radiological injury nowadays easily available. Moreover, the concept of a focus of maldeveloped tissue remaining (in some ill-defined form) and being the site of localization of infection is unacceptable in the light of increasing modern knowledge of abnormalities (hamartomata). These grow with the rest of the body and when complicated by infection still show evidence of their original character.

Ericsson and Ivemark (1958) regarded the presence of primitive ducts and/or cartilage as essential for labelling a kidney as embryologically maldeveloped. These ducts were observed in the medulla, either singly or in groups "lined with cuboidal epithelium and had concentric rings of connective tissue that seemed to contain collagen". The opinion that they were "remnants of the mesonephric duct", however, is pure speculation. Primitive ductules were presumed to be straight collecting tubules.

In the present case, it was not possible to classify the organ as a dysplasia by the features mentioned by Eisendrath (1935) nor were there the primitive ducts or "ductules" of Ericsson and Ivemark (1958). Incidentally, the 3 kidneys described by these last authors ranged in age from fifteen months to four years (when tissues of embryonic form would be expected to be found) but here the tissues had been subjected to stimuli of various kinds for twenty-five years. Tubular remnants, closely resembling the "primitive tubules" described by Marshall (1953), were found to be represented solely by persistent diverticula; microdissection showed them to be groups of diverticula, often flattened, sometimes complete cysts and, at times, atrophic remnants but not actual tubules.

Ciliated epithelial cells lining tubules in some cases have been likened to those of epididymal tubules (Cicchino, Yates and Chappell, 1951; Johnson and Anderson, 1953), but were not observed in the present case.

Even though prior lesions may be present, a renal condition may be due to postnatal inflammation. Ritter and Kramer (1950) noted that atrophic pyelonephritis was produced experimentally in guinea pigs by partial or complete ureteric obstruction. Johnson and Anderson (1953) also stressed the increased liability to renal infection following ureteral obstruction of even short duration. The evidence for long-standing pyelonephritis included leucocytic infiltration, tissue scarring, dilatation of tubules and glomerular hyalinization. In the specimen described here, there was ureteral obstruction but at what stage it originated, prior or subsequent to the renal changes, is not known.

An important feature of postnatal injuries, in addition to inflammatory changes in connective tissue, is the various degrees and forms of parenchymal regeneration. Regenerated cells are frequently of an anaplastic type and they are often wrongly regarded as being "primitive" or "embryonic". It has been commonly and uncritically assumed that a morphological similarity of regenerating and embryonic cells, which is often superficial and unsubstantial, means actual identity.

Numerous examples of regeneration are available. Tubule cell regeneration in experimentally produced uranium nephritis in guinea pigs may "alter materially the structure of the tubule" (Oliver, 1915), the new cells being heaped in masses filling the lumen. The hyperplasia of convoluted tubules in chronic nephritis is well known (Oliver, 1939) and the heterogenic growth" (Oliver, 1950) shown in diverticula and cysts is becoming recognized. In potassium depletion in rats, there is an increase in the intercalated cells in the collecting tubules (Oliver, 1944-45; Oliver *et alii*, 1957). The suggestion that these are "embryonically misplaced members of the heavily granular epithelium of the nephron tubule", however, does not take cognizance of the known capacities of postnatal cells for metaplastic change.

Regenerated cells are frequently of a "primitive" type (Oliver, 1953). In corrosive sublimate poisoning regenerating tubular epithelium may be flattened in form and the accompanying electrolyte loss has been correlated with this structural "immaturity". Phosphatase is absent from abnormal nephrons and is presumed to support the idea of a developmental anomaly in the Fanconi syndrome (Clay, Darmady and Hawkins, 1953); but, apart from other reasons for doubting the truth of this conclusion, phosphatase is absent from regenerating epithelial elements. Oliver (1953) observed that regenerating epithelial cells, following uranium nitrate poisoning, lacked mitochondrial bodies. Mitochondria are concentrations of enzymes so that such cells are both structurally and functionally atypical and, if this be accepted as an adequate criterion, "primitive". This does not imply an antenatal phenomenon or "congenital" anomaly but merely that the cells are dedifferentiated.

An outstanding feature of the present specimen is the preponderance of connective tissue. Connective tissue increases either as a "replacement" of degenerated or atrophic parenchyma or as part of the inflammatory changes following injury. It is less susceptible to injury than epithelium and proliferates more readily so that after a series of injuries connective tissue will naturally preponderate. Tilp (1912) distinguished between cases in which the "vascular connective tissue apparatus" is not disturbed and those in which the stroma is also damaged.

In the former, regeneration may be complete but in the second it is not; that is, the more severe the injury the less likely is parenchymal regeneration of a significantly functional kind. In the more severe injuries there is obliteration of the lumen by ingrowth of connective tissue; this, however, as stated, is an indication of the degree of epithelial damage; it is not due to some primary activity of the connective tissue.

Here, the essential unity of tissues must be recognized. One is the complement of another; thus, both in original development and later growth, parenchymal and connective tissue are closely related and form an integrated structure. Thus it is misleading to think of connective tissue pressing on tubules, invading them or affecting them mechanically in any way. The exceptions, where there is intact epithelium, such as neoplastic strictures of tubes and pressure of a structure on an adjacent organ, need not detain us here. The agents which produce damage usually affect parenchyma to a greater degree than connective tissue but in the absence of epithelial degeneration and atrophy replacement by connective tissue will not occur.

The more gross kind of change, occurring at intervals along tubules, is responsible for many of the blindly-ending dilated tubules; associated with this is epithelial regeneration shown both as diverticular formation and stratification of cells. Similar features were observed by Oliver and Luey (1934) in their specimens of haemorrhagic Bright's disease. The connective tissue was largely collagenous and it was almost impossible to tease out the atrophied parenchyma; this framework was, in a great majority of instances, essential for the illustration of the numerous "grape-like" clusters of diverticula which collapsed and disintegrated when separated from the binding tissue. They gave a comprehensive description of the range of hyperplastic and atrophic patterns at a late stage of renal disease. In the irregular tubules "irregularities take the form of fusiform swellings that taper to narrow necks, bulbous varices that jut out in grape-cluster fashion from the tubule, or spherical appendages". As Oliver (1939) has remarked, "we have as yet no reason to suppose that one atrophied or distorted nephron behaves differently from another whatever the cause of the regression may be".

In the case described here we have an end-picture in which the sole survivors from the

whole gamut of pathological episodes are collecting tubules and their regenerative offshoots.

The study of the present specimen by microdissection, by demonstrating the nature of some of the microscopically observed structures, proves of value in disposing of some of the arguments based on what are now demonstrably wrong premises. This applies particularly to multiple diverticula (some of which have been previously interpreted as groups of embryonic tubules). The ghosts of glomeruli indicate the previous, and probably not far distant, presence of complete nephrons; the absence of convoluted tubules conforms with their well-known susceptibility to injury in many conditions. At the same time, the tissue proliferations of various kinds demonstrate the capacity, in the most adverse conditions, for considerable, even if functionally ineffective, regeneration.

SUMMARY AND CONCLUSIONS

Microdissection of a grossly contracted kidney from a patient aged 25 years showed the remnants of tubules and ducts. No glomerulus was found but there was evidence of tubular hyperplasia, particularly in the form of diverticular formation and localized cellular proliferations. Damage had been too severe and extensive for this regeneration to significantly influence the progress of the renal degeneration and atrophy.

The usual explanation of an abnormally small kidney is that it is due to "dysplasia" or some other "congenital" process. This is based on the fundamental misconception that, in postnatal life, tissues have arrived at an immutable state. Any inflammatory processes are "explained" as to be expected because abnormal tissues are more liable than others to injuries of various kinds. Furthermore, the assumption that structures, which resemble foetal tubules, are in fact the original foetal components which have remained in this form for many years of adult life ignores the essential plasticity of tissues and implies that they are not influenced by the many stimuli which have clearly been acting on the other tissues of the organ. The postulate that such components lack the lability observed elsewhere is unreasonable.

Precise and definite evidence as to the stages and pathways in the pathogenesis of the fibrotic kidney are absent, having been lost in a maze of repeated injuries and the various reactionary and regenerative activities which follow. Conclusions must therefore be speculative. However, if the disease process began early in life and was continuous and progressive, extreme changes are to be expected.

Whatever the time (antenatal or postnatal) at which the process began, the important feature in the present case is that the parenchymal tissues of the kidney, even though practically overwhelmed by recurrent infections, still manifest the capacities of all tissues (whatever their stage in the life of the individual) for regenerative change.

ACKNOWLEDGEMENT

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THE DIAGNOSIS OF PORTAL HYPERTENSION

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Portal Pressure in Health

THERE seems to be some uncertainty about the normal portal venous pressure. A good deal of attention has been paid to its variation in disease, but of its fluctuation in the physiological range there is a striking lack of reliable recorded evidence.

It is known to rise markedly when the venous return to the heart is impeded as in the Valsalva manoeuvre. For this reason no estimate of portal pressure can be trusted if there is any hint of respiratory obstruction (Taylor, 1954).

The pressure also becomes tremendously elevated whenever there is contraction of the diaphragm as in choking or in coughing. The difference in pressure registered on a rubber balloon, placed in turn in the lower end of the oesophagus and in the fundus of the stomach may, on deep breathing, be as much as 160 cm. of water (Taylor, 1954). It is important to remember that this pressure is much in excess of that recorded during quiet respiration in patients with advanced portal hypertension. Surprisingly enough, it seems to be little affected by posture (Taylor, 1954).

Fall in the general systemic blood pressure leads to an increase in the hepatic blood flow (Culbertson *et alii*, 1951) and even when this fall is profound, as in shock, the portal pressure is reported to fall only a very little (Wiggers *et alii*, 1946; Engstrand, 1949; Boerema, 1954; Taylor, 1954).

It also varies with intra-abdominal pressure; for example, in the dog it can be elevated by distension of the bowel (Olerud, 1953) or by the less physiological manoeuvre of injecting saline into the peritoneal cavity (Bergstrand and Ekman, 1957).

When one bears in mind that, in addition to these physiological variations, no two observers seem to measure from the same base line, it should come as no surprise that portal pressure, as measured in healthy adults, varies over a wide range.

Sherlock (1955) has collected together from the literature, values for portal pressure, estimated by a variety of methods, but all in patients who were believed to be normal. The range in the whole series was from 34 to 224 mm. of water and even individual observers reported values covering almost as wide a range.

Hunt, in his Jacksonian Prize Essay (Hunt, 1958) found in 26 normal controls that the average value was 100 mm. of water and anything in the range of 100 to 130 mm. of water seems to us to be an acceptable average figure.

Portal Pressure in Disease

Although Gilbert and Carnot (1902) are believed to have been the first to use the term portal hypertension, realization of the importance of this change in the haemodynamics of the hepatic circulation came slowly and it was not until 1937 that direct measurements were first made of the pressure in the splenic vein at operation in man by Thompson and his colleagues (Thompson *et alii*, 1937). It was the work of this team at the Presbyterian Hospital in New York which first indicated clearly the significance of portal hypertension and the history of this syndrome has been largely written since their time. As in the study of high blood pressure in the systemic circulation, some arbitrary figure has to be selected as the upper limit of normal. Although there has been a considerable difference of opinion on this score, most clinicians have come to accept any reading over 300 mm. of water as being quite certainly in the hypertensive range. Some would be satisfied with any value in excess of 200.

A rise in pressure in the portal circuit may come about in a variety of ways.

It would, for example, be reasonable to assume that, in all patients with splenomegaly, the increased volume of blood contributed to the portal stream from this source might, of itself, cause a rise in portal pressure. Surprisingly enough, however, although the pressure in patients with splenomegaly and an unobstructed vein may be raised, it seldom

*Presently in receipt of a Research Grant from the Rockefeller Foundation.

reaches the high level encountered in hepatic cirrhosis (Edwards, 1955; Basu and Das, 1956).

A more likely cause would be the presence of any obstruction of the portal vein. The blood is propelled along the portal vein by a variety of mechanisms and the part that each plays is by no means completely understood. Chief among them must surely be the pumping action of the diaphragm (Alexander, 1951; Taylor, 1954) when, by its contraction it induces fluctuations in pressure both in the chest and in the abdomen. The flow is also aided by that component of the arterial pressure which is transmitted across the capillary bed in the viscera and maybe also by the peristaltic contraction of the smooth muscle of the intestine. So long as the efficiency of the pumping system remains unimpaired, the pressure in the portal vein will rise in the presence of any block to the free flow of blood from the viscera to the right side of the heart. The obstruction, in the great majority of cases, occurs in the liver as a sequel to hepatitis or to alcoholism when the regenerating liver nodules are assumed to flatten the sinusoids against the unyielding fibrous envelope of each lobule. It is, however, always a matter of some wonder how, in the presence of obstruction, such apparently weak pumping forces are able to push the pressure in the vein up to levels of 300 mm. of water or more.

Normally the relatively high pressure in the branches of the hepatic artery becomes equilibrated in the liver sinusoids with the low pressure in the portal circulation, but there is a suggestion that, in cirrhosis, this arterial pressure can, through a shunt mechanism, be more directly transmitted across the liver and lead to a rise in portal pressure to very high levels.

Herrick, over fifty years ago (Herrick, 1907) showed by liver perfusion experiments that, although in a normal liver a 40 mm. rise in the arterial perfusion pressure resulted in only a 1 mm. rise in portal pressure, in a cirrhotic liver a 26 mm. rise in arterial pressure was sufficient to produce the same result.

Portal pressure can also be considerably affected by gross ascites, although there is no reason to believe that the presence of ascites is in any way responsible for the initiation of the rise in portal pressure.

METHODS OF DIAGNOSIS

(1) *History and physical signs*

The diagnosis of portal hypertension, as in most conditions, depends in the first instance on the history and physical signs. We do not propose to recapitulate the clinical features of portal hypertension, all of which are well known.

The most frequent symptom, as well as the most urgent, is the occurrence of bleeding, either haematemesis or melaena. There will usually be an antecedent history of hepatitis or of alcoholism. In our community, urgent gastro-intestinal bleeding is most often due to a bleeding peptic ulcer (Lovell, 1958). It is not surprising, therefore, that a few patients with cirrhosis come to operation with this mistaken diagnosis, the more so since we have come to learn that cirrhosis and peptic ulceration often occur together (Markoff, 1954). A barium swallow, done as an emergency, may contribute usefully to the diagnosis and it should also be a source of comfort to remember that an enlarged spleen is the single most important physical sign in the diagnosis of portal hypertension (Sherlock, 1955). The discovery of spider naevi is also a material help in diagnosis.

The presence of a collateral circulation is, however, more valid and more convincing evidence. Although it is common enough to detect a few veins on the anterior abdominal wall in cirrhosis, a typical *caput Medusae* has not been observed in any of our patients.

Although the standard textbooks state that the development of portal systemic shunts through the haemorrhoidal plexus may manifest itself clinically as haemorrhoids, this seems in practice to be a most unusual finding (Macpherson, 1953; Taylor, 1954; Hunt, 1958). Certainly we have found no record of urgent rectal bleeding as a symptom of portal hypertension. If a high portal pressure alone were the precipitating cause of gastro-intestinal haemorrhage, bleeding should occur just as frequently at the distal as at the proximal end of the bowel.

(2) *Measurement of portal pressure*

Even when, from the history and from the physical signs, the surgeon has arrived at a confident diagnosis of portal hypertension, he is always anxious to have a reasonably exact measure of its degree, since all his surgical

efforts to date have been directed towards the control of gastro-intestinal bleeding by some porta-systemic shunting operation, the purpose of which is to lower the pressure in the portal circulation.

Portal pressure can be measured in several ways, some direct and some indirect.

Direct method

(a) By cannulation of the portal vein or a large tributary at laparotomy. The most obvious method is by introducing a saline-filled polythene tube into a reasonably direct tributary of the portal vein and connecting it up to a simple water manometer. It is important to select for cannulation one of the larger terminal tributaries since the pressure is consistently higher in the smaller and more peripheral vessels (Macpherson, 1953). Care should be taken that the polythene is not kinked or angulated. Commonly, the mid-axillary line is selected as the base line but with the abdomen opened it is easier and more accurate to select the anterior margin of the vertebrae (Taylor, 1954).

The disadvantages of measuring pressure in this way are obvious. A laparotomy is obligatory and it can seldom be justified for this cause alone. Moreover, opening the abdomen, especially under anaesthesia and with controlled respiration must alter fundamentally the changes which normally prevail above and below the diaphragm. It is, however, a convenient, simple and presumably accurate method of establishing the presence of portal hypertension when contemplating a porta-systemic shunt, or when an unsuspected cirrhosis is encountered at an emergency operation for haematemesis. It would obviously be preferable to be able to estimate portal venous pressure without the need for laparotomy and the possibilities of alternatives of this nature have been extensively explored.

(b) Cannulation of an accessible collateral vein. The measurement of the pressure in a prominent and accessible collateral vein on the abdominal wall has been shown in cirrhotics (Davidson *et alii*, 1950) to approximate closely to values established at operation. In our experience, however, it is unusual to find any worthwhile collaterals in this situation and we have never had the opportunity of exploring the usefulness of this method.

(c) There was a time when efforts were made to measure the pressure in the haemorrhoidal plexus but this, too, has never proved to be a worthwhile or indeed a practicable procedure.

(d) Allison (1951) and later Palmer (1951) were able to measure the pressure inside the varices in the submucosa of the lower oesophagus in patients with cirrhosis by puncturing them with a long needle passed through the oesophagoscope. It is claimed that a reasonably accurate measurement can be made in this way in rather less than 50 per cent. of patients. We are exceedingly doubtful of the value of this method of estimating portal pressure. It is, moreover, surprising that one can apparently carry out this manoeuvre without significant risk of inducing continuing and serious bleeding.

There being no other point at which the portal vein or its branches become accessible, the possibility was next explored of being able to measure portal pressure indirectly.

Indirect method

(a) Hepatic vein catheterization. The first effort in this direction was to measure the pressure in the hepatic vein in the belief that this value might follow in some degree pressure in the veins on the other side of the liver. A similar technique had already been worked out by Dexter and his colleagues (Hellems *et alii*, 1948) to enable him to measure the pressure changes in the pulmonary capillaries. Both the portal and hepatic veins are valveless and they function as end-veins which communicate freely with each other in the hepatic sinusoids where the pressures in these two systems (and also in the hepatic artery) are equilibrated. Friedman and Weiner (1951) showed in dogs that a catheter with an end-hole, wedged into and blocking a fine tributary of the hepatic vein gives a pressure measurement only a few cms. of water lower than that recorded when it is lying free in the portal vein. Similar studies were made about the same time by Myers and Taylor (1951) and they later extended their investigations to man.

The first authoritative work on this method of determining portal venous pressure in man came from Sherlock and her colleagues in 1953 (Paton *et alii*, 1953) and their experience has since been widely confirmed (Myhre, 1954; Reynolds *et alii*, 1957).

The technique is a reasonably simple one: it requires only the facilities for cardiac catheterization. It can be conducted on the conscious patient under mild sedation. A radio-opaque catheter is introduced into an arm vein and is threaded under fluoroscopic control through the right atrium and beyond into the inferior vena cava. The tip of the catheter is then rotated so that it enters the right hepatic vein (Fig. I). Sometimes it slips into either the renal vein or into an intercostal or other tributary. When the catheter has engaged the right hepatic vein it is then advanced for a distance of about 10 cm. until it is arrested. The catheter may be seen to kink when gentle pressure is applied and this appreciation of impaction is quite unmistakable. At this point slight tension may be necessary to disimpact it. The tip of the catheter (which has a terminal opening only) can now be assumed to be blocking a tributary of the hepatic vein. This is the "occluded" or "wedged" position. It is then connected to an electromanometer and a reading taken. The catheter is then withdrawn a little and a second reading taken in the "free position" and also in the inferior vena cava (Fig. II). It is desirable to reduce the manipulations of the catheter to the minimum and with this in view a double-lumen tube has recently been developed to make it possible to measure the occluded and the free pressures without moving the catheter.

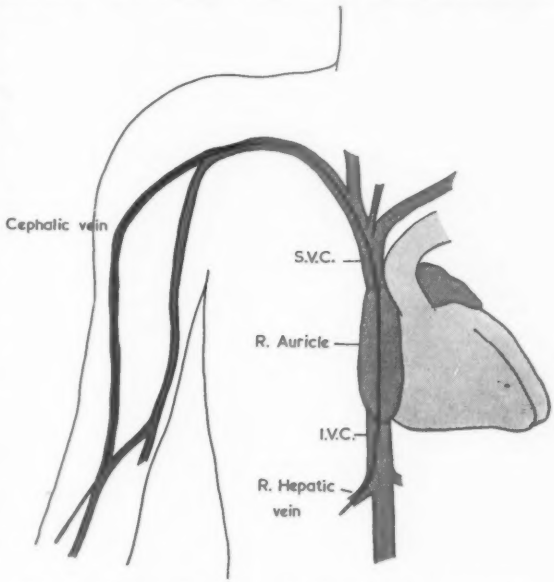

FIG. I. Catheterization of the right hepatic vein from the arm, through the superior vena cava and the right side of the heart.

Cardiac arrythmia may occur should the catheter stray into the right ventricle or into the coronary sinus. In the normal person the gradient between the "occluded" and "free" pressure is a gradual one (Fig. III). In cirrhosis on the other hand pressure in the occluded position is high and it falls sharply as the catheter is withdrawn into the free position. A high occluded pressure will also be recorded in any long-standing congestive cardiac failure, as for example, in constrictive pericarditis. In this condition, however, there will be no fall in the pressure when the catheter is manipulated into the free position; in fact, quite the reverse may hold true.

There is ample evidence in the literature that in cirrhosis the wedged pressure is only a little lower than the portal venous pressure as determined directly at operation (Krook, 1953; Reynolds *et alii*, 1955). This was not, however, the experience of Ekman (1957) who found the wedged pressure to be a little lower than the portal venous pressure in 5 patients out of 11. We have been able on several occasions to demonstrate to our own satisfaction that there is no major discrepancy between the two. The intra-sinusoidal pressure presumably lies between the portal pressure and the wedged hepatic pressure. Most observers take the occluded hepatic pressure as an index of portal vein pressure. Some workers, however (Reynolds *et alii*, 1955) believe that the use of an external zero reference point favours inconsistency between observers. They suggest that the difference between portal and caval pressure gives the best measure of the resistance to the flow of blood through the liver.

Unfortunately, there must always be a measure of risk in passing a catheter through the right side of the heart. Arrythmia may occur should the catheter stray into the ventricle or, worse still, into the coronary

sinus. For this reason one of us (G.B.) has developed the technique of introducing the catheter into the hepatic vein from below by threading it up into the inferior vena cava from the saphenous vein. The angle between the inferior vena cava and hepatic vein is an acute one but it has now been successfully negotiated on several occasion (Figs. IV and V).

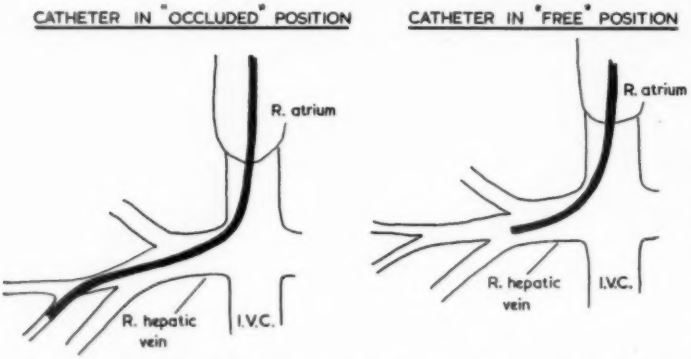

FIG. II. The catheter, which has a terminal opening, is shown in the "occluded" and "free" positions.

(b) The second indirect method of measuring portal pressure is by measurement of the pressure in the splenic pulp. The red pulp of the spleen communicates directly with the primary venous channels which represent the beginnings of the splenic vein. It would seem for this reason probable that the pressure in the splenic pulp would be related to that in the portal vein and this has been shown to be the case (Lebon *et alii*, 1954). As might be anticipated, the pressure in the splenic pulp is slightly higher than the portal pressure as measured in a tributary at laparotomy but the difference is seldom more than 30 mm. of water. The difference between the two is significantly greater if there is any ascites (Ekman, 1957). It is, therefore, important to tap ascites before taking an intra-splenic pulp pressure. We have had the opportunity in several patients of recording intra-splenic pulp pressure and wedged hepatic pressure concurrently. The two values have, in the majority, corresponded quite closely (Figs. VI and VII and Table 1). The relative heights of pressure in the splenic pulp and in the occluded hepatic vein may also make it possible to determine not only the presence of a block but also its site. If, for example, the occluded hepatic vein pressure is normal and the intra-splenic pressure is raised, there is every likelihood that the block is extra-hepatic.

The measurement of intra-splenic pulp pressure in this way is a reasonably safe procedure, always provided a fine needle is used and there are enough platelets and a satisfactory prothrombin level. It is possible that the use of the Seldinger technique may lessen the risk considerably (Seldinger, 1957). The needle is introduced either through the overlying chest wall or directly through the abdominal parietes. It is obviously easier to enter an enlarged spleen. Local or general anaesthesia can be used so long as the patient can be induced to breathe quietly while the needle is in position.

FIG. III. Tracing to show how, in a healthy subject, there is a fall in the hepatic vein pressure as the catheter is withdrawn from the "occluded" to the "free" position.

Having introduced a needle percutaneously into the spleen for a pressure measurement it is customary to inject 50 ml. of a radio-opaque dye (50 per cent. Diodine) in rather less than 10 seconds. Radiographs are taken serially during the injection.

In a normal person the dye can be followed through the splenic and portal veins and into its distributaries in the liver. There will, however, be no filling of any of the tributaries of the portal vein despite the fact that the dye may be forced into the splenic pulp under very considerable pressure.

In the presence of portal obstruction the dye by-passes the liver and returns to the heart through collateral vessels. The most constant of them is a leash of veins which passes towards the cardia (Fig VIII). Occasionally, the inferior mesenteric vein will be outlined or veins passing along the falciform ligament to the anterior abdominal wall. We have not yet a wide enough experience in this field to interpret profitably the pattern of the intrahepatic vessels in cirrhosis.

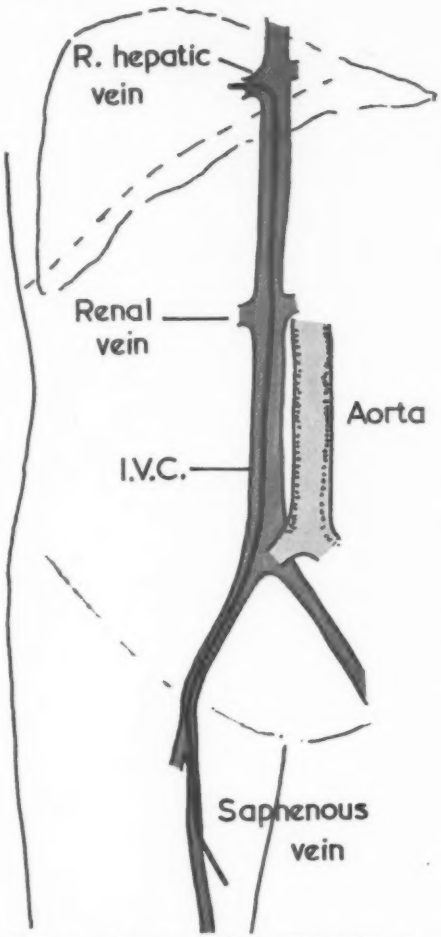

FIG. IV. Diagram to illustrate catheterization of the hepatic vein from the thigh.

It is now widely accepted that the presence of collaterals in a spleno-portagram done percutaneously is an index of portal hypertension although it is equally true that a raised pressure may sometimes be found in patients with a normal X-ray (Atkinson *et alii*, 1955; Basu and Das, 1956). In other words, the degree of collateral circulation is not necessarily a measure of the severity of the portal hypertension (Ekman, 1957). This is certainly true in acute cases and where there is a profusion of collaterals (Sherlock and Walker, 1957). Moreover, in an extrahepatic block there may be big collaterals without a raised pressure (Hunt, 1958). In the main, however, the demonstration of collaterals on venography by splenic puncture is good evidence of hypertension. It is, however, important to remember that a venogram made at operation by injecting the dye into a jejunal or omental tributary may cause, in a normal person, some regurgitation into one of the large tributaries such as the inferior mesenteric and does not for this reason afford nearly such valid evidence. This is, however, the method favoured by Hunt and his colleagues (Hunt, 1958).

TABLE 1

SIMULTANEOUS "OCCLUDED" HEPATIC VEIN PRESSURES AND SPLENIC PULP PRESSURES IN A GROUP OF CIRRHOTIC PATIENTS

Age	*Sex*	*Portal Pressure (130 mm Water)*	*Splenic-Pulp Pressure (143 mm Water)*
62	F	338	325
57	M	247	234
66	M	260	273
34	M	208	182
54	M	182	130
27	M	156	156

The normal values are shown in parentheses.

There are, of course, some risks associated with this manoeuvre of trans-splenic portal venography. The dye may be injected under the capsule of the spleen or into the peritoneal cavity or even into the stomach. Fortunately, none of these happenings seems to cause serious trouble. However, for obvious reasons, we prefer to postpone this investigation until immediately before operation.

(c) If the presence of a collateral circulation is an index of portal hypertension then varices demonstrated in the oesophagus by

barium swallow should afford equally valuable evidence. It is, however, a much less certain way of demonstrating varices than either oesophagoscopy or spleno-portography.

It is true that use of the Valsalva manoeuvre not only inhibits peristalsis and relaxes the oesophagus but also raised the pressure in the veins in the submucosal plexus and so makes them more obvious (Nelson, 1957). The chance of demonstrating varices is also heightened by screening with the patient in the prone position with increased intra-abdominal pressure obtained by a pillow under the obdomen and the Valsalva manoeuvre (Luke, 1958). It is, however, not always easy to make a firm diagnosis radiologically of the presence of varices and this affects its usefulness as a source of evidence. The investigation can, however, fortunately be carried out without much upset to the patient. From our experience, however, the radiologist very often has great difficulty in making a firm diagnosis of the presence of varices. It is now believed that varices are shown more often and more convincingly by oesophagoscopy or, alternatively, by splenoportal venography. This is all the more un-

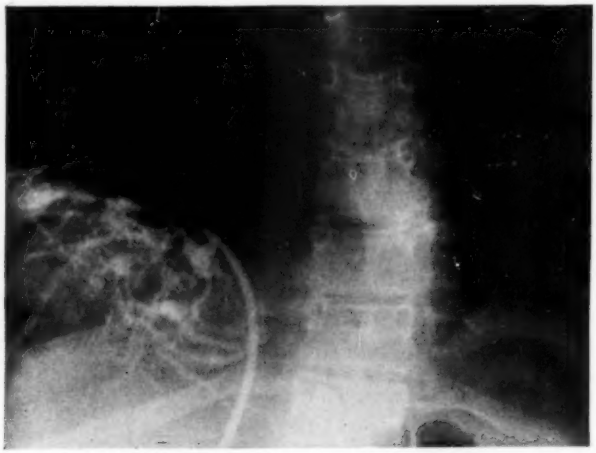

FIG. V. Catheterization of the right hepatic vein via the right long saphenous vein. Contrast medium has been injected retrograde into the hepatic vein and has outlined the veins in the right lobe of the liver.

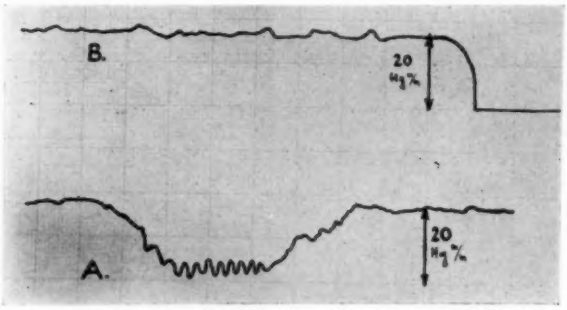

FIG. VI. Simultaneous records of "occluded" hepatic vein pressure (A) and splenic pulp pressure (B) in a patient with advanced cirrhosis. During the phase of low pressure in tracing (A) the catheter was in the inferior vena cava and registered the pressure changes in the right side of the heart.

fortunate when one recalls how little a patient is upset by barium swallow.

COMMENT

From work in dogs, in which cirrhosis has been produced by carbon tetrachloride and alcohol intoxication over a long period it is believed that measurement of portal pressure is the single most sensitive index of liver damage. We have indicated that there are several ways in which the pressure can be measured, some direct and some indirect. Although all of them can be practised without undue risk, none is such a harmless manoeuvre that it can legitimately be used repeatedly to follow the progress of liver disease in any single individual. For this reason we have little satisfactory documentary evidence of the natural history of cirrhosis in terms of the change in the haemodynamics of the liver circulation to which it gives effect and this is a deficiency which seems to us to

FIG. VII. Percutaneous trans-splenic portal venogram with a catheter in the right hepatic vein.

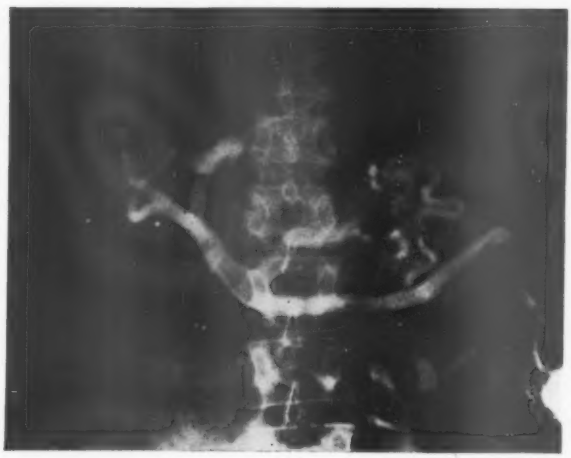

FIG. VIII. Percutaneous trans-splenic portal venogram demonstrating the presence of collaterals in the region of the cardia.

limit very significantly our understanding of the unsolved problems of cirrhosis.

It is, moreover, important to realize that, having determined the portal pressure, one cannot claim to have established an exact measure of the threat of urgent haemorrhage. It is towards the protection of our patient from just this risk that our surgical endeavours have, up to the present, been mainly directed and although by some shunting procedure we may lower significantly the portal pressure, we achieve this only by a further and maybe disastrous depletion of the blood supply to the liver parenchyma.

SUMMARY

A review has been made in the light of our experience of the methods used to establish the diagnosis of portal hypertension.

We have had enthusiastic support in our enquiries from Dr. H. A. Luke, Director of Radiology of the Alfred Hospital. We would like to record our thanks to our colleagues in the many hospitals who have allowed us to investigate patients under their care.

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CONTRACTION IN WOUNDS MADE AT SHORT TIME INTERVALS

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Introduction

SINCE the early work of Carrel (1913) when he demonstrated the effect of embryonic tissue juices in accelerating the rate of growth of connective tissue there has been evidence produced both for and against the presence of substances liberated from a healing wound, which are capable of affecting the rate of closure of a wound made at a later date.

FIG. I. Photograph showing the rectangular wound on the right flank of an animal twenty-four hours after wounding.

There is little doubt that if a wound is reopened a few days after being made and then resutured, there is an accelerated rate of gain in tensile strength on the second occasion (Savlov and Dunphy 1954). However there is no agreement concerning the effect of a primary wound on a second made subsequently in another part of the body. Arey (1936) has reviewed the literature to that date and since then more rapid closure of the second wound has been reported by Young, Fisher and Young (1941). Taffel, Donovan and Lapinski (1951), on the other hand, found that trauma from a large soft tissue wound on the back did not affect the rate of healing of a stomach wound (judged by bursting pressure) made five to fifteen days after the primary wound. Similarly Billingham and Russell (1956) could detect no difference in the specific rate of contraction in wounds made eight to seventeen days apart. The importance of adequate controls was demonstrated by Sandblom and Muren (1954) and they attributed the earlier observed acceleration of the second wound to differences in skin temperature following depilation.

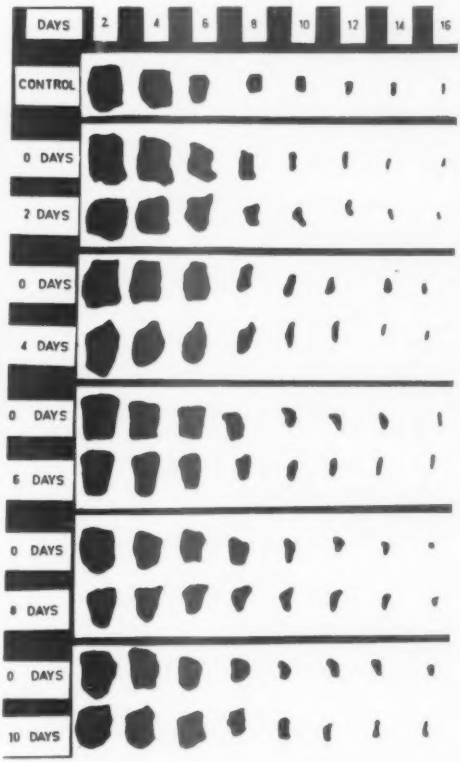

FIG. II. Representative tracings from individual animals of each group showing the similarity of the wounds on the two sides to the control animal.

Present Experiment

Method

Wounds two cm. square were made down to the deep fascia in the right flank (Fig. I) of four control rats (100-150 gm) and the rate of healing followed by tracings every

second day on to celluloid strips. The tracings were then projected by means of a photographic enlarger and the image measured by radial planimetry and the average results graphed.

Four animals were used for each observation and the results averaged and graphed. Dressings were not used and infection was uncommon; two animals were excluded from the results for this reason.

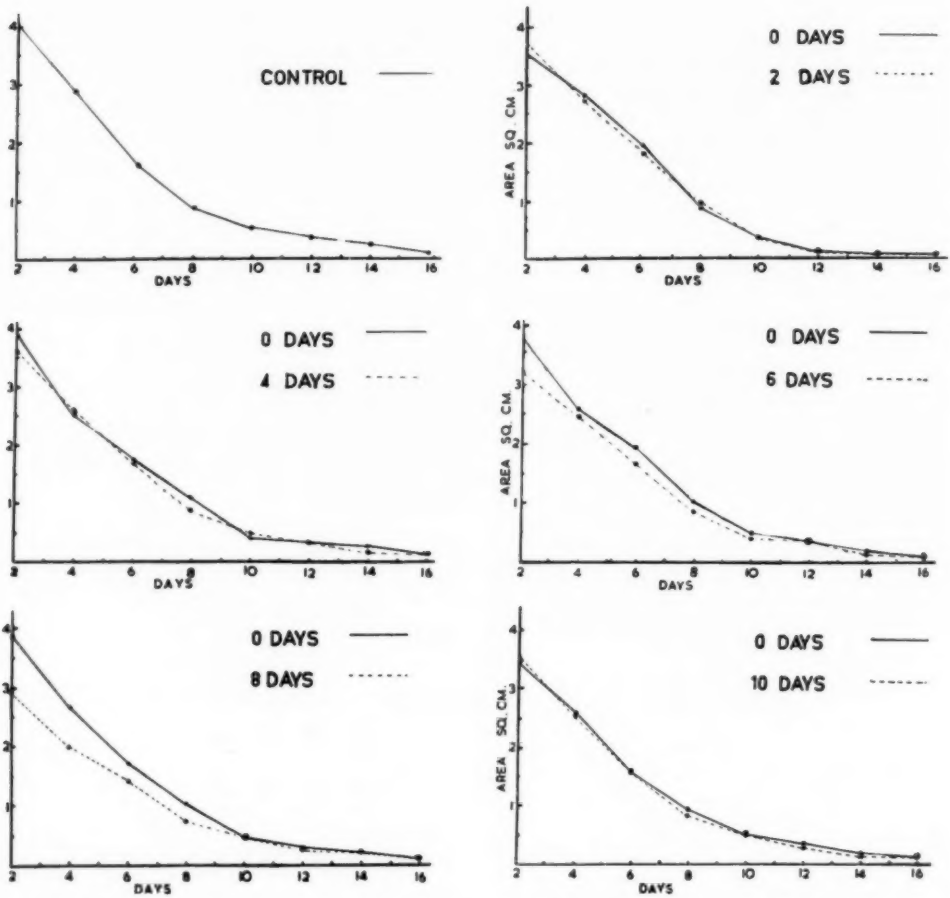

FIG. III. Graphs of the average rate of closure of control and staged wounds showing no significant difference between primary and secondary defects. It will be noted that wounds made on the eighth day were slightly smaller but healed in the same time.

A similar wound was then made in a further twenty animals and the effect of a further wound made on the opposite side on the second, fourth, sixth, eighth and tenth days was studied with respect to the original wound and to the rate of closure of the second wound.

Results

A representative set of tracings from each group is shown in Fig. II. The average areas have been graphed and it is obvious (Fig. III) that there is no significant difference in the rate of closure of the two wounds.

In the group in which the secondary wound was made eight days after the primary wound, the secondary wound (due to a technical error) was made slightly smaller. It is of interest that the average rate of healing was slightly slower than in the larger control wound, so that they both healed in the same time.

DISCUSSION

This experiment supports the view that no substance, demonstrable by present techniques, is liberated into the blood stream from a healing wound which can significantly alter the rate of closure of another wound made subsequently in another part of the body.

The experiments of Young *et alii* (1941) can be criticized because the end point of the experiment was taken as the day when the scab finally separated and the rate of closure was expressed as the time for this to occur. It is extremely difficult to decide, within a day or so, when a wound has healed and it would seem more reasonable to take the slope of a graph, obtained by serial observations, as the measure of the rate of closure. Billingham and Russell (1956) used rather long time intervals (eight to seventeen days) and although they carefully measured the diminishing area and concluded that there was no difference on the two sides, one would not have expected any difference at this stage. It is most probable that if there were any demonstrable effect it would be manifest during the first five days, for it is during this period that the bulk of the wound bed is laid down. In the present experiments no such difference was observed and it is concluded that each separate wound stimulates its own tissue response which is essentially local and closure proceeds autonomously.

The presence of substances liberated from healing wounds has on occasions been held to explain the accelerated rate of growth of metastases following operative removal of a primary tumour. It can be stated that the present experimental evidence does not support this contention and therefore this phenomenon must be explained on other grounds.

SUMMARY

Large skin wounds were made at staged time intervals on the opposite flanks of rats. No evidence was found for the presence of substances liberated from the primary wound which influenced the rate of closure of a subsequent wound.

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A MODIFIED TECHNIQUE OF LOWER LIMB VENOGRAPHY

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VENOGRAPHY of the lower limb was introduced by Dos Santos in 1938, although he was not the first person to inject radio-opaque substances into the vascular system.

Since 1938 many methods of venography have been described, but none of them has proved fully satisfactory. A venogram may be performed to demonstrate the state of the deep veins, the number and adequacy of the valves of the deep system, or the presence of incompetent communicating veins. The method to be described enables these points to be demonstrated.

Three methods of performing venography are described in the current literature:—

(a) Descending or functional venography with injection into superficial veins (Moore, 1955).

(b) Intra-osseus venography (Lester and Lampe, 1957; Dodd and Mistry, 1958).

(c) Ascending venography (Gryspeerdt, 1953; Dodd and Cockett, 1956).

Descending venography has not received widespread acceptance, as it is of limited application.

The intra-osseus technique of lower limb venography, at present under trial in a number of centres, would seem to have serious disadvantages. General anaesthesia is necessary, as the injection of radio-opaque material into the bone is painful, and a failure in asepsis can have disastrous consequences. Lester and Lampe (1957) suggest that the intra-osseus technique should be reserved for patients without suitable subcutaneous veins.

Dow (1951) investigated various methods previously described in the literature, and his work provided the basis of a method of ascending venography suitable for the study of the deep veins of the lower limb. The aim was to drive the contrast medium from the superficial veins into the deep veins of the leg, injection being given into a superficial vein of the foot after a tourniquet had been applied to the lower leg immediately above the malleoli. The tourniquet had to be placed sufficiently low to avoid obscuring the lower tibial region, and to be tight enough to occlude only the superficial veins. It had previously been found that injection into the veins of the foot when the superficial veins of the leg were not obstructed by a tourniquet gave inadequate filling of the deep venous system. The use of a tourniquet has become accepted as a part of the technique of ascending venography (Gryspeerdt, 1953). To be of value, a venogram should give an undisturbed picture of the venous system and it is suggested that the use of a tourniquet distorts

FIG. I. The right hand photograph shows an X-ray taken at the completion of the injection of 60 cc. of 30 per cent. Urografin, when the dye has filled numerous superficial and deep veins. The photograph on the left is of an X-ray photograph taken after the patient has performed the Valsalva manoeuvre for ten seconds and shows an increased density of dye in the incompetent communicating vein (arrow).

the natural channels of flow, causing false appearances. In this article is described a minor modification of technique which avoids the use of a tourniquet and at the same time allows adequate filling of the deep veins.

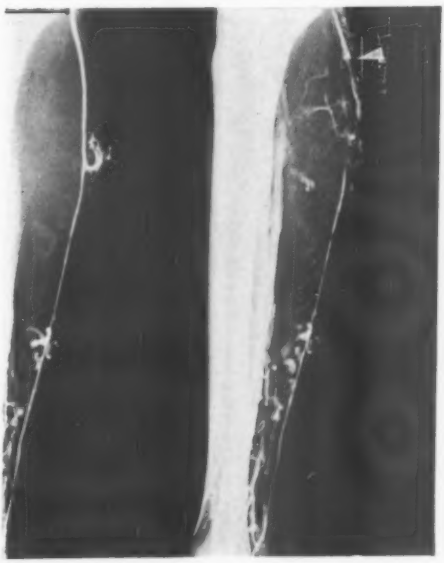

FIG. II. Ascending venogram showing filling of superficial channels on performing the Valsalva manoeuvre. The long saphenous vein has a competent valve (arrow) just below knee level. This illustrates the increased filling of the superficial veins from the deep, which occurs due to the physiological block to the venous return with the Valsalva manoeuvre. No tourniquet is necessary.

Gryspeerdt (1953), whose method has been used as the basis of this investigation, describes the use of the Valsalva manoeuvre to demonstrate the valves of the deep venous system. The manoeuvre is of additional value in that it provides useful information about incompetent communicating veins. Cockett and Dodd (1956) describe a "bleed-back" test used at operation to demonstrate reversal of blood flow through a communicating vein with an incompetent valve. Normally the blood flows from the superficial veins towards the deep veins, a reversal of flow being prevented by a valve near the junction of the communicating vein with the deep system. If the superficial end of the exposed communicating vein is clamped and the vein is cut deep to the forceps, active bleeding will only take place if the valve has been destroyed. This reversal of blood flow may be demonstrated radiologically. The deep veins are filled with contrast medium and an X-ray photograph is taken. The patient performs the Valsalva manoeuvre and a second X-ray photograph is taken. The increased filling of the superficial veins occurs in the vicinity of an incompetent communicating vein, and the incompetent vein itself is more clearly outlined (Figs. I and II).

FIG. III. Injection of dye into a superficial vein of the foot in a proximal direction, without the use of a tourniquet, has filled the long saphenous vein and its tributaries without filling the deep veins.

METHOD

The patient lies supine on the table with the feet tilted 10° downwards. The leg to be examined is placed in slight external rotation, with a plastic foam pad beneath the heel. The table is fitted with a mobile Potter-Bucky tray. The patient rehearses the Valsalva manoeouvre and the skin of the foot is cleaned with a mild antiseptic and isolated by sterile towelling. A vein on the medial side of the foot below the medial malleolus is selected with care, and an injection of 0.5 per cent. Xylocaine is made into the adjacent subcutaneous tissues. After exposing the vein a

fine polythene cannula is inserted in a distal direction as far as it will go, i.e. towards the sole of the foot.

The cannula is connected to a three-way tap, and isotonic saline is allowed to drip through the cannula from a raised bottle.

No tourniquet is used.

An injection of 60 cc. of 30 per cent. Urografin is delivered slowly from a 100 cc. syringe connected to the tap with a Luer-Lock connection, the approximate time of injection being one minute.

FIG. IV. The dye from the previous injection was cleared from the leg by the saline infusion and elevation of the foot. The polythene cannula was withdrawn and re-inserted into the distal limb of the same vein. A second injection of dye was given in a distal direction thirty minutes later, without the use of a tourniquet, and good filling of the deep venous system occurred.

A radiograph is taken at the completion of the injection and the patient asked to perform the Valsalva manoeuvre for ten seconds. A second exposure is made and the Potter-Bucky tray quickly moved so that an X-ray photograph is made of the popliteal region while the patient is still performing the Valsalva manoeuvre (this does not require more than a further five seconds). The patient relaxes and a fourth radiograph is made of the femoral and iliac region a minute later. The saline infusion is continued while the films are developed.

The vein is ligated with fine catgut and the incision closed with a single mersilk suture. A firm dressing is applied, including an elastic bandage, and early ambulation is encouraged.

FIG. V. A venogram of a leg of an elderly woman, who had a chronic venous ulcer on the inner aspect of her leg for thirteen years. Contrast medium has been injected through a polythene cannula in a distal direction and shows adequate filling of both the deep and superficial veins as high as the upper third of thigh, without the use of a tourniquet.

COMMENT

The injection in a distal direction makes the use of a tourniquet unnecessary. The contrast medium passes through innumerable communicating veins of the foot into the deep system of the leg. A small quantity of the dye passes directly up the superficial veins, but the bulk of the injected material ascends through the deep venous system. When the patient performs the Valsalva manoeuvre, some of the dye is forced out into the subcutaneous veins by reversal of flow along communicating veins with incompetent valves (Figs. III and IV).

The interpretations of the venograms in this series were confirmed by subfascial dissection (Figs. V and VI).

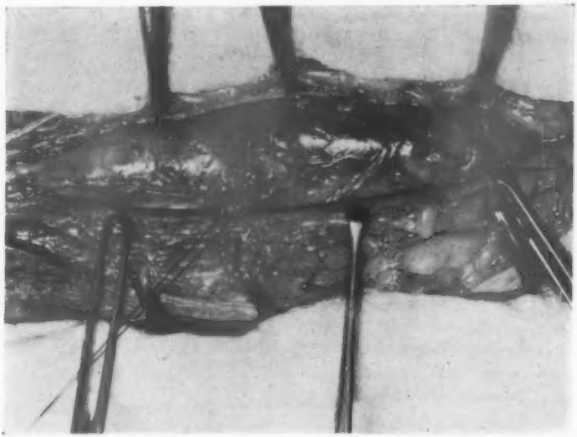

FIG. VI. A photograph of the subfascial dissection of the communicating veins illustrated in Fig. V. The Allis forceps are on the deep fascia and thread ligatures encircle the veins immediately beneath the fascia (arrowed).

Summary

Two modifications are suggested for the accepted method of performing an intravenous ascending venogram. The first modification is the injection of the contrast medium through a fine polythene cannula inserted into a small vein of the "ankle flare" below the medial malleolus, in a distal direction, the use of a tourniquet being unnecessary. Secondly, it is suggested that the Valsalva manoeuvre is of value in demonstrating reversal of blood flow through communicating veins which have an incompetent valve. A technique is described incorporating the modifications with illustrations of their use.

Acknowledgement

I wish to acknowledge the valuable assistance given by Dr. B. Vaughan and his department in the development of this technique and the interpretation of the X-ray photographs.

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TRAUMATIC RUPTURE OF THE KIDNEY IN CHRISTMAS DISEASE

By Robert Shannon

Brisbane

CHRISTMAS disease was first described in 1952 by Biggs and others, and derives its name from the first patient in whom it was diagnosed. The clinical picture of Christmas disease is indistinguishable from haemophilia. Both conditions are inherited as sex-linked recessive characters, but it would appear that the defect in Christmas disease may not be completely recessive and cases of Christmas disease have been reported in females (Hardisty, 1957). The bleeding-time, tourniquet test, platelet count and prothrombin times are all within the respective normal ranges in both conditions, while each typically shows a prolonged whole blood clotting-time and deficient consumption of prothrombin during clotting.

The fundamental defect in both haemophilia and Christmas disease is failure to form intrinsic thromboplastin normally. In the case of haemophilia this is due to lack of antihaemophilic globulin, which is present in the fibrinogen fraction of normal plasma. In Christmas disease, the plasma contains the normal amount of antihaemophilic globulin and lacks a different factor normally found in serum, namely, the Christmas factor, which is present in the beta-globulin fraction of normal plasma.

Although Christmas disease is not very common, its recognition in any one patient is important because the treatment is not the same as that for haemophilia. While both will respond equally to treatment with normal plasma, Christmas disease will not respond to the therapeutic administration of purified antihaemophilic globulin. Even if normal plasma is transfused an important difference is observed. The classical antihaemophilic globulin disappears within a few days after the transfusion of 500 cc. of normal plasma, but the stability of the beta-globulin, which corrects Christmas disease, is such that a favourable influence on blood clotting can be observed as long as three weeks after the transfusion. Also, the life of antihaemophilic globulin in stored blood is short, whereas the Christmas factor is moderately well preserved, in bank blood stored at 4°C. Brafield and Case (1957) state that there is good preservation of both antihaemophilic globulin and Christmas factor if fresh plasma is immediately frozen and stored at –20°C.

Case Report

R.M., a 36-year-old single male, was admitted to the Brisbane Hospital on 23rd April, 1957, shortly after being involved in a car accident. He could not remember the accident clearly and was not sure if he lost consciousness. On admission, he was conscious and orientated and complained of headache, left sided abdominal pain and left shoulder pain.

Physical examination revealed a well-developed male in some distress. Blood pressure was 110/75 mm. of mercury and pulse rate 108 per minute. His abdomen was tender and guarded in the left upper quadrant, and he was tender in the left loin. Blood was present in his urine.

Past history

The patient stated that he had been a bleeder as long as he could remember. The first severe episode was at the age of nine or ten, when he bled after teeth extraction. His first blood transfusion was at the age of fourteen years. Since then, he has had recurrent episodes of bleeding, sometimes after trauma, but also spontaneous bleeds into the knee joints and from the renal and gastro-intestinal tracts, requiring blood transfusions on many occasions. No abnormality of the gastro-intestinal tract has ever been demonstrated on barium meal examination.

On previous admissions, when his blood disease had been fully investigated, his bleeding time was within normal limits, the clotting time prolonged (7 to 9 minutes), platelet count normal, and prothrombin time slightly prolonged. The prolongation of prothrombin time was shown to be due to a weak anticoagulant and not to factor VII deficiency. In the thromboplastin generation test the patient's serum was shown to be defective and as there was no factor VII deficiency this indicated that the patient had Christmas disease.

Family history

He was the only child of his father. His mother re-married and had fourteen children by her second husband. Three of his five step-brothers are confirmed Christmas disease sufferers and one of these was involved in the same accident. This step-brother had suffered a head injury but was conscious and orientated on admission. However, his condition deteriorated and a right subdural haematoma was evacuated twenty-four hours later. The patient died

fifteen hours after operation, and at post-mortem a large subdural haematoma was found to have reformed.

A nephew, a son of one of R.M.'s step-sisters, had recently been in the Brisbane Children's Hospital with bleeding tendencies. An uncle is a bleeder and two male cousins have bled to death, one after a tonsil operation and the other after an accident. Several male cousins of his mother are bleeders.

His mother, two step-sisters and one female cousin bruise and bleed easily, but have not been fully investigated. Firkin (1958) states that carriers of Christmas disease often give a history of a greater tendency to bleed than normal and he has detected a diminution of Christmas factor in the serum of carriers.

As the patient was obviously not going to survive with continued conservative management, operation was performed on the 14th day after injury.

The kidney was exposed through a lumbar incision with removal of the left 12th rib. A large amount of dark clotted blood surrounded the left kidney in the retroperitoneal space, extending to the midline anteriorly and from the diaphragm to the pelvis. The capsule of the convex border of the kidney had been stripped up and ruptured, but no large laceration of renal cortex was present. Clot was present in the renal pelvis. The kidney was removed, a considerable amount of clot evacuated and after careful haemostasis, the wound closed around a drain to the kidney bed. Gel-foam was packed into the renal space and into the wound as it was closed.

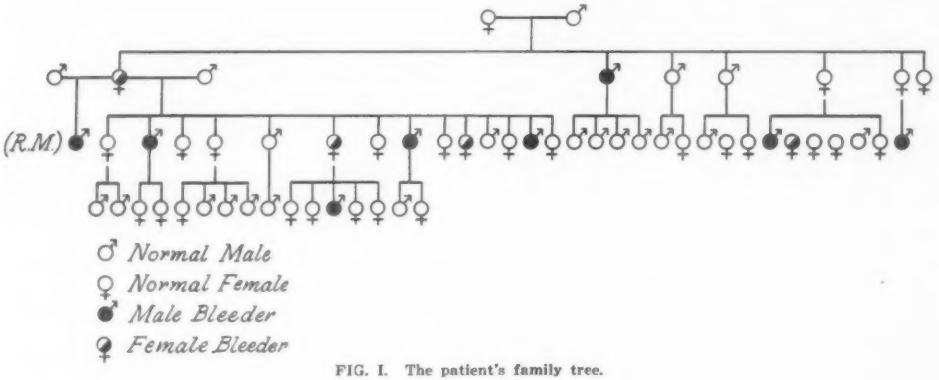

FIG. I. The patient's family tree.

Progress

The day after admission, marked bruising and swelling of eyelids and forehead appeared, and increased for some days. Haematuria continued and a mass became palpable in the left side. Elsewhere, the abdomen was soft. Three days after injury the patient began to experience severe left-sided, colicky pain, presumably clot colic, and this continued intermittently until operation.

An intravenous pyelogram done six days after injury showed a normal right kidney, but poor concentration by the left kidney.

Blood transfusions of one litre were given on the 2nd, 8th, 11th and 12th days after injury, but bleeding from the left kidney continued, as indicated by continued haematuria and clot colic, an increasing swelling in the left loin and a falling haemoglobin percentage.

Bleeding and clotting times were within normal limits on several occasions, but this was after transfusion, which could be expected to bring the clotting time down to normal values.

By the 10th day after injury the swelling in the left side extended from the costal margin to inguinal ligament, and almost to the midline anteriorly. The size of the mass was causing respiratory embarrassment and the patient became increasingly dyspnoeic.

On the day of operation, a transfusion of 1½ litres of blood was given, and ½ to 1 litre on the next three days, and then every two or three days as required, for the next fortnight. Very little fresh oozing occurred till the 10th post-operative day, when there was a fair amount, but after adequate blood transfusion (3 litres) on consecutive days this diminished, and thereafter old clot continued to discharge for about three months. Abdominal distension was troublesome in the early post-operative period, but this subsided with gastric suction. The patient was finally discharged four months after admission with his wound almost healed. He had been back at work as a panel beater for some months but recently had another gastro-intestinal bleed requiring transfusion.

DISCUSSION

Extensive surgical procedures on haemophiliacs have been successful in areas where local measures could be applied to control blood loss, but the mortality following internal surgery in established cases of haemophilia is relatively high, probably 50 to 60 per cent. (Craddock *et alii*, 1948).

In the case of internal surgery, recovery depends on adequate transfusion before, during, and after operation to maintain an adequate level of the deficient factor. It would seem that the greater stability of the Christmas factor makes surgery safer in these patients than in true haemophiliacs.

In the case reported, surgery became necessary because of the increasing size of the mass, which was causing respiratory embarrassment. Perhaps the quantity and freshness of the blood transfused was inadequate to provide sufficient of the missing factor to ensure coagulation and the success of the initial conservative management. At operation, a large amount of clot was removed, but care was taken not to disturb the periphery of the haematoma which may have caused fresh bleeding in an inaccessible region. Fresh post-operative bleeding was not really a problem but frequent blood transfusions were given in the early post-operative period as a prophylactic measure.

Throughout the management of this case bank blood stored at 4°C was used for transfusion. Recent investigation has shown that Christmas factor is more stable at –20°C and Brafield and Case (1957) now recommend fresh serum stored at this temperature for the treatment of Christmas disease.

SUMMARY

Christmas disease is clinically indistinguishable from haemophilia.

The Christmas factor is more stable than the deficient factor in haemophilia, as a result of which Christmas disease may be treated with stored bank blood, but this should not be used in haemophilia.

A case of traumatic rupture of the kidney in Christmas disease is reported with recovery following nephrectomy.

ACKNOWLEDGEMENT

I wish to thank Dr. A. D. D. Pye, General Medical Superintendent of the Brisbane Hospital, for permission to publish this case record.

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Books Reviewed

CARCINOMA OF THE LUNG.

Edited by J. R. BIGNALL, M.D., M.R.C.P. Edinburgh and London: E. & S. Livingstone Ltd., 1958. 10" x 7", xii plus 298 pp., 62 figures, various tables. Price: 55s. (stg.).

This book is one of the series of monographs on neoplasms at individual sites. It is a most comprehensive exposition of present knowledge of all aspects of lung carcinoma by a group of eminent contributors from London. A short historical introduction precedes sections on mortality statistics, causation, pathology, course of the disease and treatment by surgery, radiotherapy and chemotherapy, each beautifully presented by an author already famous for his work in his particular field. The section on surgical treatment is written by W. P. Cleland. Each section is followed by a long list of references.

There is nothing to criticize in this book. The standard throughout is excellent and the printing, binding and illustrations are equally good. It promises to be the standard work on the subject for some time to come.

ANATOMY AND PHYSIOLOGY FOR NURSES.

By W. P. GOWLAND, M.D., F.R.C.S., and JOHN CAIRNEY, D.Sc., M.D., F.R.A.C.S. Fifth Edition. New Zealand: N. M. Peryer Ltd., 1958. 8¼" x 5½", 376 pp., 204 figures. Price: 45s. net.

This standard and excellent textbook so well known has been completely re-written in this, the fifth edition.

Its presentation is most attractive, the larger and newer type makes for easier reading and study.

The drawings in black and white are clear, distinct and in most cases, self-explanatory.

The authors have combined their particular knowledge, viz. of anatomy and physiology, to produce an integrated study of each system of the body, giving the student nurse a picture of the structure and function at the same time.

The chapters on biochemistry and microscopic anatomy are necessary introductions to such a scheme.

The authors have not only combined their knowledge of anatomy and physiology; but have applied it to medicine and surgery, e.g. the note on obstructive jaundice and its treatment with vitamin K; artificial respiration; and the brief but excellent note on hydatid cysts.

This applied anatomy and physiology, introduced throughout the book, surely must stimulate study and make anatomy and physiology more interesting to the nurse, who has no opportunity to study these two subjects in the practical laboratories.

This scheme is in line with the new nursing curriculum which provides that anatomy and physiology be taught simultaneously with medicine and surgery.

The chapters on the minute structure and the functions of the nervous system and the sympathetic nervous system are very comprehensive and complete; but most probably a little too difficult for the first year nursing students to assimilate; however they are excellent chapters for reference in her future studies.

There is an excellent chapter on nutrition and diet which should be studied and learnt by every nurse.

An added chapter at the conclusion of the book on the distribution of the spinal nerves to the limbs is a most valuable addition for the use of physiotherapists.

One can have nothing but praise for this excellent book which every nurse must possess.

THE FOREQUARTER AMPUTATION.

By H. F. MOSELEY, M.A., D.M., M.Ch.(Oxon.), F.R.C.S.(Engl. and Canada), F.A.C.S. Edinburgh: E. & S. Livingstone Ltd., 1957. 11" x 9", viii plus 79 pp., 11 plates (in colour), 25 figures. Price: 42s. stg.

In style, clarity and form it is difficult to imagine a finer example of the printer's art than exhibited in the production of this monograph. To the publishers, Messrs. E. & S. Livingston Ltd., therefore, we give full marks. Superbly coloured plates, adequately "flagged," iliustrate the text at appropriate places. Indeed one wonders why black and white photographs of dissections, even though accompanied by adequate diagrammatic keys, are included at all. The contrast is too marked.

As to the context — it is, as the author states in the preface, a collection of previously published material into one small volume, to which has been added lessons in surgical anatomy and some historical aspects of absorbing interest. These include a brief resume of the published details of traumatic forequarter amputations. On reading these we are saddened at the number of child workers overtaken by this disaster in days when conditions of work were both more rigorous and less safe than those enjoyed today.

Berger's classical forequarter amputation technique is faithfully described and, since few surgeons perform the operation other than very occasionally, the monograph provides an easy and convenient preoperative "run over the course." One can imagine occasions when the posterior approach of Littlewood, next described, may seem more convenient. By this technique ligation of the subclavian vessels is virtually the penultimate procedure, and, as Moseley states, "they are at a considerable depth when the operation is performed from the posterior aspect." This would seem to be a disadvantage since most of us, when called upon to perform forequarter amputation never feel really happy until these vessels are safely secured.

The indications for forequarter amputation are concisely stated, and short chapters on scapulectomy, interscapulo-thoracic resection, and post-operative rehabilitation are included. A very complete bibliography completes an excellent, useful and convenient publication.

Book Received

STAPHYLOCOCCAL INFECTIONS.

By IAN MACLEAN SMITH. Chicago: Year Book Publishers Inc., 1958. 7¾" x 5¼", 180 pp. Price: £2 6s. 9d.

Royal Australasian College of Surgeons

Proceedings

At a meeting of the Council of the College held on the 20th and 21st February, 1959, the following matters of general interest were dealt with:—

Final Fellowship Examination, October, 1958

It was reported to the Council that at the October, 1958, Final Fellowship Examination thirty-four candidates presented for the full examination in General Surgery and the Special branches of Surgery, of whom twenty-four were approved. A temporary modified type of Final Examination was held in Auckland and in Melbourne and of the seventeen candidates who presented sixteen were approved.

The following candidates have been admitted to Fellowship of the College:—

Dennis Deane Arnold, John Robert Badger, John Sadler Barnett, Peter John Blaxland, Alec Graeme Bond, John Gwynne Brockis, Roderick Ronald Collmann, William Sydney Egerton, Richard Dyason Ferris, Paul Joseph Fogarty, Richard Peter Freeman, Ronald Edward Gristwood, Bruce Macffarlane Hay, John Keith Henderson, Patrick Fraser Howden, Desmond Garvan Hurley, Nicholas Nikolaevich Iovetz-Tereshchenko, Brian Maurice Johns, Kevin Johns, Paul Garner Large, George Hector Levien, Desmond David McGuckin, William Richard McKechnie, Ian Lumsden McVey, Roland William Donald Middleton, John Stanley James Morley, James Kevin O'Reilly, Iven Alastair Page, Colin Gordon Paull, Vernon Glyde Renowden, Leith Alexander Riddell, Edmond Graham Roberts, Walter Henry Collins Scott, Harman Gilbert Smith, Robert Darlow Smith, Olaf McClure Spence, Geoffrey William Sprigge, Norman Leslie Stokoe, Stanley Henry Watson, David Zufi.

Final Fellowship Examination for the Faculty of Anaesthetists, October, 1958

Nine candidates presented for the Final Examination for Fellowship of the Faculty of Anaesthetists held in October, 1958, of whom five were approved.

The following candidates have been admitted to Fellowship of the Faculty following the examination:—

Leon Richard Alfred Bryan, Max Adderly Griffith, Allan Metcalfe Hall, Brian Thomas Jordan, Patrick Alan Maplestone.

General Scientific Meeting, 1959

The programme for the General Scientific Meeting to be held in Sydney was arranged. The meeting will commence on Tuesday, 18th August, 1959. Throughout this day surgical films will be shown with commentaries. The Inaugural Meeting will be held in the Great Hall of the University of Sydney on the evening of Tuesday, 18th August, 1959. Sir Alexander Murphy, M.C., M.D., F.R.C.P., F.R.A.C.P., will deliver the George Adlington Syme Oration, entitled "Barbers, Surgeons and Tradition."

The diagnosis, pathology and treatment of Cancer occupies the mornings of Wednesday, 19th, and Friday, 21st August, 1959. At various sessions, the subjects of multiple cancers, chemotherapy, cytological diagnosis and adrenalectomy will be discussed. Special attention will be given to upper alimentary kidney and soft tissue tumours.

Symposia have been arranged on Arteriography in Modern Surgery, the present position of Surgical Tuberculosis, and discussions on Intravenous Cholangiography, the place of Tracheotomy and Deaths under Anaesthesia. Thursday morning will be devoted to short papers on scientific subjects, and in the afternoon there will be scientific exhibits at the Medical School, King George V Hospital, Royal North Shore Hospital, and the special unit of the New South Wales State Cancer Council for the investigation and treatment of cancer.

There will also be sectional meetings for the various specialties and of the Faculty of Anaesthetists.

Social events will include a Fellows' Dinner and a theatre party for the wives of Fellows.

The New South Wales State Committee would be greatly helped if Fellows could notify their intention of being present at the meeting and advise whether they will be accompanied by their wives and also indicate what functions they would like to attend. Communications should be addressed to the Honorary Secretary of the New South Wales State Committee, P. J. Kenny, at 149 Macquarie Street, Sydney.

Specialist Surgeons

The Council considered various submissions from State Committees concerning the definition of a Specialist Surgeon and as a result formulated the following formal definition as being practicable for the present:—

1. A Specialist Surgeon is a legally qualified medical practitioner who is either a Fellow of the Royal Australasian College of Surgeons or in the possession of a qualification recognized by that College.
2. Such Specialist Surgeons should be recorded on a National Register of Approved Specialists.
3. For a limited period, the specialist qualifications of medical practitioners based on practical experience rather than academic qualifications should be the subject of individual consideration for admission to the Register of Approved Specialists.

Representation of the College on the electoral bodies of teaching hospitals

The Council seeks representation for the College on the electoral bodies of teaching hospitals. It was noted with satisfaction that following representations made to the Government of South Australia the *Hospitals Act* in South Australia has been amended giving the College and the Royal Australasian College of Physicians representation on the electoral bodies of both teaching hospitals in Adelaide.

Alan Newton Prize, 1958

The Alan Newton Prize for 1958 was awarded to G. R. Stirling. The subject of the essay was "The Surgery of Congenital Malformations of the Heart and Great Vessels."

Alan Newton Prize, 1960

The subject set for the essay for the Alan Newton Prize for 1960 is "The Pathology and Surgery of Hernias of the Abdominal Wall."

Gifts to the College

The Council desires to acknowledge with gratitude the following gifts:—

1. A silver porringer, from the estate of the late Sir Thomas Dunhill.
   This porringer was presented to Sir Thomas Dunhill by Her Majesty, the late Queen Mary, and is accompanied by her personal note.
2. The late Sir Thomas Dunhill's insignia of G.C.V.O. and C.M.G., together with his service and commemoration medals.
3. Mrs. Ivan Jose presented a portrait of her husband, by Ivor Hele, which has been hung in the Council Room.
4. Mrs. D. J. Glissan has endowed a prize in memory of her husband, the late D. J. Glissan, a Foundation Fellow of the College. The prize will be awarded biennially to a Fellow of the College who publishes a paper on an orthopaedic subject in the *Australian and New Zealand Journal of Surgery* which is considered to be the best paper published in the previous two years.

Sims Commonwealth Travelling Professor, 1959

W. C. Gissane visited Australasia as Sims Commonwealth Travelling Professor for 1959 and during the period of his visit an Honorary Fellowship of the College was bestowed upon him.

Sims Commonwealth Travelling Professor, 1958

B. K. Rank was welcomed home at the Council meeting following his tour of Canada as Sims Commonwealth Travelling Professor for 1958.

Development Funds (Australia and New Zealand)

The Development Fund (Australia) now stands at £12,130 and the Development Fund (New Zealand) is in credit to the extend of £259.

The Council wishes to acknowledge the receipt of the following further donations to these two funds:—

AUSTRALIA

M. S. Alexander	£10	0	0
D. Beard	5	5	0
R. Beard	10	10	0
G. M. Bedbrook	10	0	0
K. C. Bradley	20	0	0
D. O. Brown	20	0	0
F. J. Clark	20	0	0
*C. L. Coghlan	27	0	0
J. L. Connell	20	0	0
Dr. T. P. Crankshaw	5	5	0
*R. A. Craven	20	0	0
D. O. Crompton	20	0	0
V. H. Cumberland	20	0	0
Dr. L. G. B. Cumpston	2	2	0
N. M. Cuthbert	21	0	0
Dr. E. G. Dahlenburg (per B. K. Rank)	26	5	0
*H. D. Drury	20	0	0
*H. G. Earnshaw	20	0	0
P. B. English	5	5	0
*A. W. Farmer	10	0	0
*F. R. Fay	5	5	0
H. G. Furnell	10	10	0
W. N. Gilmour	20	0	0
*E. H. Goulston	20	0	0
P. J. F. Grant	10	10	0
*E. J. Green	20	0	0
*G. S. Gunter	40	0	0
A. R. Hamilton	20	0	0
I. Hamilton	50	0	0
R. G. S. Harris	20	0	0
*E. A. E. Hedberg	20	0	0
*H. M. Jay and J. G. Jay	30	0	0
D. S. Kidd	10	10	0
E. W. Kyle	20	0	0
D. G. Love	20	0	0
*C. D. Langton	20	0	0
*T. B. Law	10	0	0
*A. H. Lendon	20	0	0
G. A. Lloyd	100	3	0
J. Loewenthal	10	0	0
W. M. C. Macdonald	10	0	0
I. H. McConchie	40	0	0
C. C. McKellar	5	5	0
A. McSweeny	10	10	0
*R. D. Marshall	10	0	0
*G. Newman Morris	20	0	0
Sir Henry Newland	20	0	0
Estate of P. L. O'Halloran	16	19	5
W. J. Pannell	20	0	0
Dr. G. Pern	5	5	0
K. W. Priddis	20	0	0
C. A. M. Renou	105	0	0
N. H. Robinson	10	10	0
W. R. Rogerson	10	0	0
*T. F. Rose	20	0	0
M. Sheppard	10	10	0
D. A. A. Simpson	5	0	0
J. A. Simpson	20	0	0
A. Daly Smith	20	0	0
J. A. Snell	10	0	0
J. W. Spence	105	0	0
*R. H. Stanistreet	10	10	0
W. Stening	20	0	0
*G. Swinburne	20	0	0
E. R. W. Thomson	20	0	0
B. F. Venner	10	0	0
R. Webb	10	10	0
E. F. West	20	0	0
*Dr. D. R. C. Wilson	5	5	0
*N. P. Wilson	21	0	0
C. J. Windsor	2	0	0
H. M. J. Windsor	20	0	0
D. Yeates	20	0	0
G. Young	50	0	0

NEW ZEALAND

W. A. Fairclough	10	10	0

*Additional donations.

Repayment of Gordon Craig Scholarships

Former Gordon Craig Scholars have now refunded an amount of £748 to the Gordon Craig Scholarship Fund. From this sum, a grant-in-aid for a special research project has been made to W. G. Lucas, F.R.C.S., who is working in the Department of Surgery at the University of Sydney.

The Council desires to thank the following additional former Gordon Craig Scholars who have made refunds to the Gordon Craig Scholarship Fund:—

D. L. Dey
W. L. Keller
I. H. McConchie
G. G. C. McKenzie
T. E. Wilson

Fellows are urged to maintain their efforts to increase both the Development Funds and the Gordon Craig Scholarship Fund as these funds are not yet large enough to enable important College work to be undertaken.

Conferment of Honorary Fellowship

On the afternoon of the 18th February, 1959, an Honorary Fellowship was conferred on the Rt. Hon. Viscount Nuffield. The ceremony took place in the College lecture hall before a large company of Fellows and guests.

In presenting Lord Nuffield, E. L. Button, a Vice-President of the College, said:—

"Mr. President,

I have the honour to present to you the Right Honourable Viscount Nuffield, Knight Grand Cross of the Most Excellent Order of the British Empire, Companion of Honour, Fellow of the Royal Society.

Lord Nuffield is already well known to this College and its friends. His life makes a success story without equal within the Commonwealth during the twentieth century. He has made the name of Nuffield synonymous with progress and generosity.

We understand that in his earlier years Lord Nuffield had some desire to train in medicine. It is thus interesting to speculate on what epoch making contribution to surgery could have accrued, if the same talents which he applied with such success in industry, had been applied to surgical practice in one or other of its various branches. Though he has not practised actively with us his contributions to surgery have been such that he has done much to make British Surgical Practice what it is today.

We think of the Nuffield College of Surgical Science which he has established with our sister College in Lincoln's Inn Fields, whereby our younger graduates can live and work in close proximity to the library, the museum, and the laboratories of the College and at the same time have the opportunity to meet and learn from their senior colleagues in the round of everyday life. We think of the Nuffield Chairs of Surgery and of Anaesthesia established in Oxford and we think of the many Nuffield Scholarships which have helped many graduates within our Commonwealth.

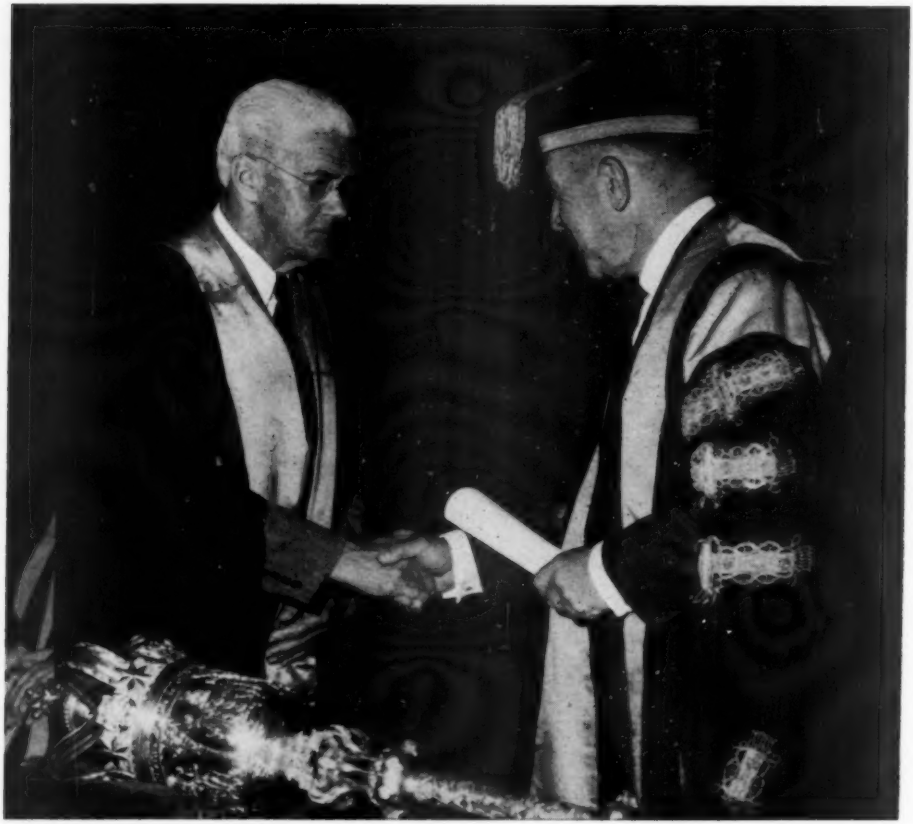
The foresight and planning behind these magnificent bequests have helped to put on a sound foundation the basic training of many surgeons coming from different parts of the Commonwealth. They have helped our own graduates coming from Australia and New Zealand to bring back to these our countries the special knoweldge which enables us to maintain our technical standards.

Lord Nuffield's contribution to training within our profession has been recognized in many ways. He holds honorary degrees from many Universities within the United Kingdom and throughout the Commonwealth. He is an Honorary Fellow of the Royal College of Surgeons of England and has the added distinction of having been awarded the Gold Medal of that College.

An Honorary Fellowship is the highest honour that this, our Royal Australasian College of Surgeons can bestow on a distinguished citizen and a distinguished colleague. Thus, Mr. President, it is a privilege and a pleasure to present to you the Right Honourable Viscount Nuffield to receive at your hands the Honorary Fellowship of our College."

After conferring the Honorary Fellowship on Lord Nuffield, the President conveyed the congratulation and good wishes of all Fellows of the College and also the congratulations of the Prime Minister of Australia, the Rt. Hon. R. G. Menzies, together with an expression of the Prime Minister's regret at being unable to be present at the ceremony. The President stated that these sentiments of happiness at the event would be felt by all people who had at heart the advancement of the science and art of Surgery towards the fulfilment of its highest ideals.

In reply, Lord Nuffield expressed his great pleasure at the honour conferred on him and spoke of his early desire to study medicine, a desire which was frustrated by the necessity of leaving school in order to support his parents. "And so I had to make motor cars."



Primary Fellowship Examination, March, 1959

A Primary Examination for Fellowship of the College was conducted in Melbourne in March, 1959; twenty-seven candidates presented for the examination, of whom eight were approved.

At the same time, a Primary Examination for Fellowship of the Faculty of Anaesthetists was also conducted. Of the seventeen candidates who presented for this examination, four were approved.

Primary Fellowship Examination, Singapore, March/April, 1959

By arrangement with the Government of Singapore, the College conducted its Primary Examination in that city in March/April, 1959. On this occasion, the examination was not conducted under the terms of the Colombo Plan. Twenty-five candidates entered for the examination, of whom six were approved.

DOUGLAS MILLER,
President.

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